CLINICAL EVALUATION AND THERAPEUTIC OUTCOME IN HIRSUTISM

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CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL EVALUATION AND THERAPEUTIC OUTCOME IN HIRSUTISM''** is a bonafide work done by **Dr. R. Subha**, at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in Dermatology, Venereology and Leprology (Branch-XII A) under my guidance and supervision during the academic year 2009 -2012.

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DECLARATION

I, **DR. R.SUBHA,** solemnly declare that dissertation titled, "**CLINICAL EVALUATION AND THERAPEUTIC OUTCOME IN HIRSUTISM"** is a bonafide work done by me at Department of Dermatology and Leprosy, Madras Medical College, Chennai-3 during the period of October 2009 to September 2011 under the supervision of **Prof. DR.S.JAYAKUMAR, M.D., D.D.,** Professor and HOD, The Department of Dermatology and Leprosy, Madras Medical College, Chennai. The dissertation is submitted to Tamilnadu Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.D. Degree (Branch-XII A) in DERMATOLOGY, VENEREOLOGY AND LEPROLOGY**.

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CONTENTS

INTRODUCTION

Hirsutism is defined as the presence of terminal coarse hairs in women in a male like distribution. Hirsutism affects approximately 5 to 10% of women of reproductive age.[1] Androgen dependent areas include the chin, upper lip, chest, abdomen, back and anterior thighs.

Hirsutism usually represents a variation of normal hair growth, but rarely it is a harbinger of a serious underlying condition. It may be a complaint for which the patient seeks medical advice or may be only a suspicion by a dermatologist seeing a woman for other androgen related problems like female pattern hair loss. Studies of the psychological burden of hirsutism suggest that it has a significant impact and adversely affects quality of life.[2]

Virilisation refers to a condition in which androgen levels are sufficiently high to cause additional signs and symptoms such as deepening of voice, breast atrophy, increased muscle bulk, clitoromegaly and increased libido. Virilisation is an ominous sign that suggests the possibility of an ovarian or adrenal neoplasm. Genetic factors and ethnic background also influence hair growth. In general, dark haired individuals tend to be more hirsute than fair individuals.

REVIEW OF LITERATURE

Perception of hirsutism is by definition subjective and women present with a wide variation in severity.[3,4] Both the severity of the hirsutism and the degree of its acceptance are dependant on racial, cultural and social factors.

Influence of hormone on hair development:

Growth of sexual hair is dependant on sex hormones, which can act on the dermal papilla to convert small, straight, non pigmented vellus hair into coarse, pigmented terminal hair. It increases the duration of the anagen phase of the hair growth cycle in the beard, axillary and pubic areas. Conversely, androgens can also have the opposite effect on specific scalp areas often in the same individual, causing balding. This involves a reverse transformation of large, terminal, heavily pigmented scalp hairs to miniaturized vellus follicles resulting in androgenetic alopecia. These contrasts must be due to intrinsic differences in gene expression within follicles at different sites.

In the skin, testosterone is converted to the more potent dihydrotestosterone (DHT) by the enzyme 5alpha reductase.[5] There are two isoenzymes of 5alpha reductase: type 2 is found in the prostate gland

and in hair follicle whereas type 1 is found primarily in sebaceous glands.[6]

In non androgen dependent areas such as the forehead and cheeks, androgens enlarge the sebaceous glands; however hair follicles remain vellus in nature. Hair growth on the chest, upper abdomen and back requires greater levels of androgens and is therefore more characteristic of the pattern typically seen in males.

In men, over 95% of testosterone originates from the Leydig cells of testes. In women, 40-50% of testosterone arises from the ovaries and adrenals while the remaining 50-60% comes from peripheral conversion of androstenedione which arises from both ovaries and adrenal glands. It can either be converted to estrogen by the enzyme aromatase in granulosa cell or to testosterone by the enzyme 17beta hydroxysteroid dehydrogenase in theca cell. In both genders, dihydroxytestosterone arises from peripheral conversion of dehydroepiandrosterone sulphate.

Pathogenesis of Hirsutism:

Hirsutism results from either an exogenous or endogenous increase in circulating androgens or from increased sensitivity of the hair follicle to normal serum androgen levels (end organ dysfunction). Although

androgen excess underlies most cases of hirsutism, the clinical severity of hirsutism does not always correlate with the expected levels of circulating androgens. There is considerable heterogeneity of responses among androgen dependant follicles in different individuals. This is due to variability in end organ sensitivity.[7,8,9]

Key androgens that may be secreted in excess include testosterone which usually originates from the ovary; DHEAS which is of adrenal origin; and androstenedione which originates from either the ovary or the adrenal gland.[10]

Prostate specific antigen measurements have a potential use in monitoring the treatment of hirsutism in addition to being a marker of androgen excess.[11]

Etiologies of Hirsutism:[12]

1. Constitutional or idiopathic hirsutism
2. Pituitary or hypophyseal hirsutism
3. Ovarian hirsutism -

- Tumoural: Arrhenoblastomas, hilus cell tumour, granulosa cell tumour, gonadoblastomas.
- Non tumoural:Polycystic ovarian syndrome, ovarian hyperthecosis.

4. Adrenal hirsutism-

- Tumoural: Virilising adenomas, carcinomas
- Non tumoural : Congenital hyperplasia, Cushings syndrome

5. Iatrogenic hirsutism
6. Hepatic hirsutism
7. Hirsutism due to alteration of peripheral conversion of androgen

Idiopathic Hirsutism:

Androgen excess is not detectable in every patient with hirsutism. Those patients with hirsutism, regular ovulation, and normal to slightly elevated androgen levels in the absence of features that suggest other causes of hirsutism are labeled as having idiopathic hirsutism.[13,14] Women with this syndrome may also present with seborrhoea, acne and alopecia (SAHA syndrome). Pathophysiology of idiopathic hirsutism is related to increased peripheral activity of 5alpha reductase in the hair follicle, which converts testosterone to the more potent androgen, dihydrotestosterone(DHT). Gonadotropin releasing hormone (GnRH) analogue and ACTH stimulation tests may uncover occult ovarian or adrenal functional hyperandrogenism in a large number of these patients. Increased prevalence of insulin resistance and impaired glucose tolerance has been observed in idiopathic hirsutism.[15]

Polycystic ovarian syndrome:

Polycystic ovarian syndrome (PCOS), also known as Stein-Leventhal syndrome, is the most common cause of hirsutism.[16] It can present with hyperandrogenism; menstrual irregularities including oligomenorrhoea, amenorrhoea and infertility; and dysmetabolic syndrome that includes impaired glucose tolerance, hyperlipidemia and obesity (Body mass index>30kg/m^2).[17] Among hirsutes with abnormal cycles, obese women have higher levels of total testosterone, free testosterone and androstenedione. These patients are also at increased risk for cardiovascular disease.[18] Polycystic ovaries are not necessary to make the diagnosis.

Proposed diagnostic criteria for polycystic ovarian syndrome[19] (Rotterdam Criteria):

Two of the three criteria necessary for diagnosis -

1. Oligo or anovulation (<8menses/year or cycles >35 days)

2. Clinical and/or biochemical signs of hyperandrogenism

3. Presence of polycystic ovaries and absence of other etiologies (Cushing's syndrome, congenital adrenal hyperplasia, androgen secreting tumors)

The gene for PCOS is inherited in an autosomal dominant pattern. Nearly half of the sisters of PCOS patients have an elevated plasma testosterone level, of which half of them (about 25% of the total) have symptomatic PCOS. Male relatives also carry the gene. Significant male balding before the age 30 years with or without concomitant insulin resistance are suggestive of male PCOS carrier status.[20] Women with polycystic ovaries and their brothers with early balding show links to the steroid metabolism gene, CYP17.[21]

The major source of hyperandrogenemia in PCOS appears to be gonadotropin dependant functional ovarian hyperandrogenism.[10] In a small fraction of patients with PCOS, elevated DHEAS is found. The high intra ovarian androgen concentration causes maturation arrest of ovarian follicles, which accounts for the “polycystic” appearance of the ovaries.

Adrenal hirsutism:

Congenital adrenal hyperplasia:

A deficiency of 21α hydroxylase is responsible for 95% of all cases.[22] It results in defective conversion of 17 hydroxy progesterone to 11 deoxycortisol. There are three clinical forms:[12]

1. Classic form or salt losing form- relate to failure of aldosterone synthesis.

2. Non classic form- accompanied by failure of cortisol synthesis. The absence of cortisol causes hyperproduction of ACTH, which produces adrenal hyperplasia. Oligomenorrhoea, clitoromegaly, masculinisation, changes in voice and lack of breast development are other manifestations.

3. Late onset congenital adrenal hyperplasia - due to partial deficiency of 21α hydroxylase which manifests when demand for steroid increases. Patients often present with premature pubarche with onset of hirsutism in the prepubertal years. Menstrual irregularity, oligomenorrhoea or primary amenorrhoea may also be present.[23]

Benign and malignant androgen secreting tumours should be suspected in cases with sudden onset and rapidly progressing hirsutism, features of virilization (deepened voice, clitoromegaly, increased muscle mass, increased libido) and significantly elevated androgen level.

Pituitary hirsutism:

Many of the endocrinopathies are the result of a pituitary adenoma. Visual field defect can occur due to impingement on the optic chiasma. Pituitary adenomas result in hyperprolactinemia, acromegaly and Cushing's disease and hirsutism. Some of the features include:

- Cushing's disease : Hypertension, proximal muscle atrophy, striae, thinned skin with easy bruising, fat redistribution (moon facies, dorsocervical fat pad, supraclavicular fat pad, central and less commonly generalized obesity), hyperpigmentation, cutaneous fungal and bacterial infections
- Acromegaly: Coarse facies, large hands, acanthosis nigricans
- Thyroid dysfunction: Tremors, brittle hair, diffuse telogen hair loss, dry skin, goiter
- Hyperprolactinemia: Spontaneous or expressible galactorrhoea

Drugs causing hirsutism:[24]

Androgenic medications:

- Testosterone
- Danazol
- ACTH

- Metyrapone
- Phenothiazine

Anabolic steroids

Androgenic progestins

- Levonorgestrel
- Norgestrel
- Norethindrone

Acetazolamide

Valproic acid

Nonandrogenic medications

- Cyclosporine
- Phenytoin
- Diazoxide
- Minoxidil
- Minocyclin
- High dose glucocorticoids
- Hexachlorobenzene
- Penicillamine
- Psoralens

HAIR - AN syndrome:

Hyperandrogenism, insulin resistance and acanthosis nigricans (HAIR-AN) syndrome is distinct from PCOS. It tends to have a greater degree of associated morbidity, like type 2 diabetes, hypertension and cardiovascular disease. The pathogenesis is thought to be related to an insulin receptor and/or post receptor defect resulting in the compensatory increase in circulating insulin and luteinizing hormone. These hormones stimulate excess ovarian androgen secretion. Associated features include PCOS, cystic mastitis, obesity, and infertility.[25]

Hirsutism in postmenopausal:

Hirsutism may be due to a number of causes including tumours. It may also be due to ovarian hyperthecosis in which ovarian interstitial cells differentiate into steroidogenically active luteinized stromal cells. Ovarian estrogen production decreases causing a reduction in concentration of SHBG. Consequently, there is an increase in the proportion of unbound testosterone, resulting in hirsutism.

Hirsutism in pregnancy:

It is due to luteoma of pregnancy, hyperreactio luteinalis or aromatase deficiency in the fetus. The maternal hyperandrogenism resolves in the postpartum period.

Hepatic hirsutism:[12]

A low level of sex hormone binding globulin (SHBG) leads to a larger amount of free testosterone and the conversion to dihydroxy testosterone may be greater. As SHBG is produced in liver, in cases of liver diseases SHBG level are decreased.

Cutaneous associations:[6]

- Acne vulgaris
- Androgenetic alopecia
- Acanthosis nigricans
- Skin tag
- Seborrhoea

Metabolic syndrome and hirsutism:

The metabolic syndrome and polycystic ovarian syndrome (PCOS) are frequently associated with abdominal obesity, dyslipidemia, and insulin resistance. Both are risk factors for cardiovascular events and stroke.[26] It is thought that hyperinsulinemia may play an important role in the development of both syndromes.[27] Thus, there is considerable overlap between PCOS and metabolic syndrome patient populations. Hirsutism in women is an important presenting clinical symptom which often leads to

the diagnosis of PCOS. Hence it is important to consider the diagnosis, implications, and treatment of metabolic syndrome in patients presenting with hirsutism.[28]

The International Diabetes Federation (IDF) definition of metabolic syndrome.

Central obesity :	Waist circumference Men >90cm Women >80cm

The above plus two of the following four features

Glucose or insulin abnormalities-	Fasting plasma glucose >100mg/dl Or previously diagnosed type 2 diabetes
Dyslipidemia -	Triglycerides >150mg/dl or specific treatment for this abnormality
Dyslipidemia -	HDL <40 mg/dl (men) HDL <50 mg/dl (women) or specific treatment for this abnormality
Elevated blood pressure -	BP > 130/85 mmHg or treatment of previously diagnosed hypertension

Clinical assessment:

Assessment of hirsutism includes the age of onset, rate of progression of hair growth and associated symptoms and signs (e.g., acne). Growth is usually slow but progressive. Sudden development and

rapid progression of hirsutism suggests the possibility of an androgen secreting tumour, in which case virilisation may also be present.

The age of onset of menstrual cycle and the pattern of the menstrual cycle should be noted; irregular cycles from the time of menarche are more likely to result from ovarian rather than adrenal androgen excess. Associated symptoms such as galactorrhoea should prompt evaluation for hyperprolactinemia and hypothyroidism. Hypertension, striae, easy bruising, centripetal weight gain and weakness suggest hypercortisolism (Cushing's syndrome). A family history of infertility and/or hirsutism may indicate disorders such as non classic CAH.

Physical examination should include measurement of height, weight and calculation of body mass index (BMI). A BMI of >25 kg/m2 is indicative of excess weight for height, and values >30kg/m2 are often seen in association with hirsutism. Notation should be made of blood pressure, as adrenal cause may be associated with hypertension.

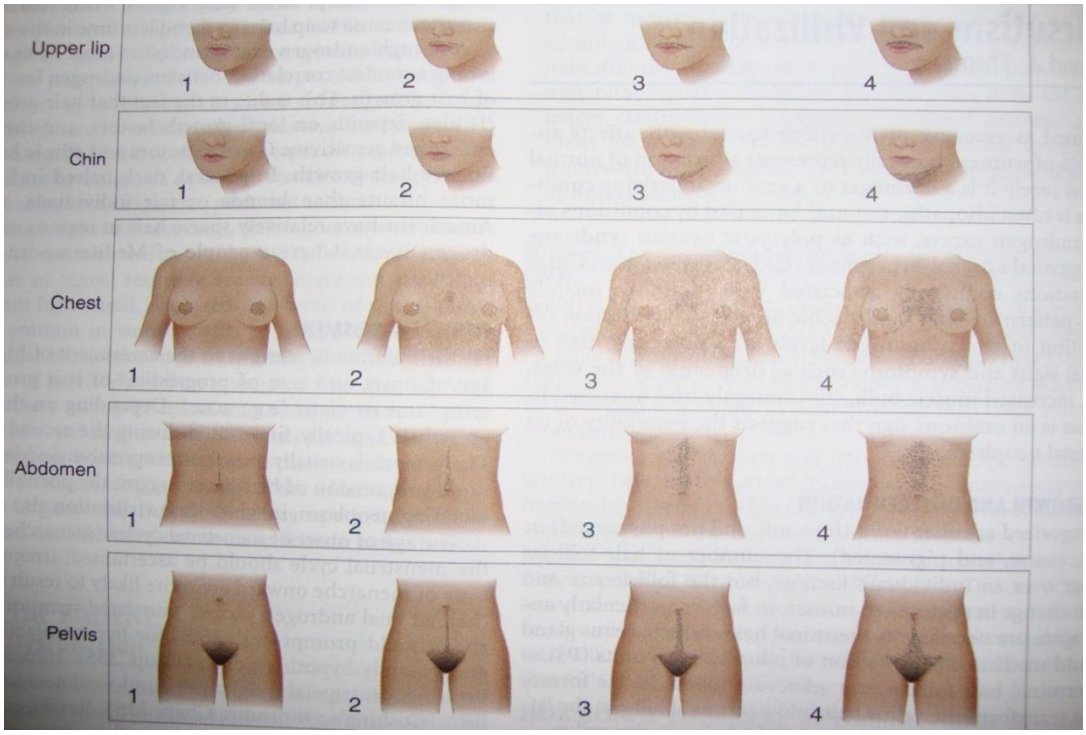
Objective measurement of hirsutism:

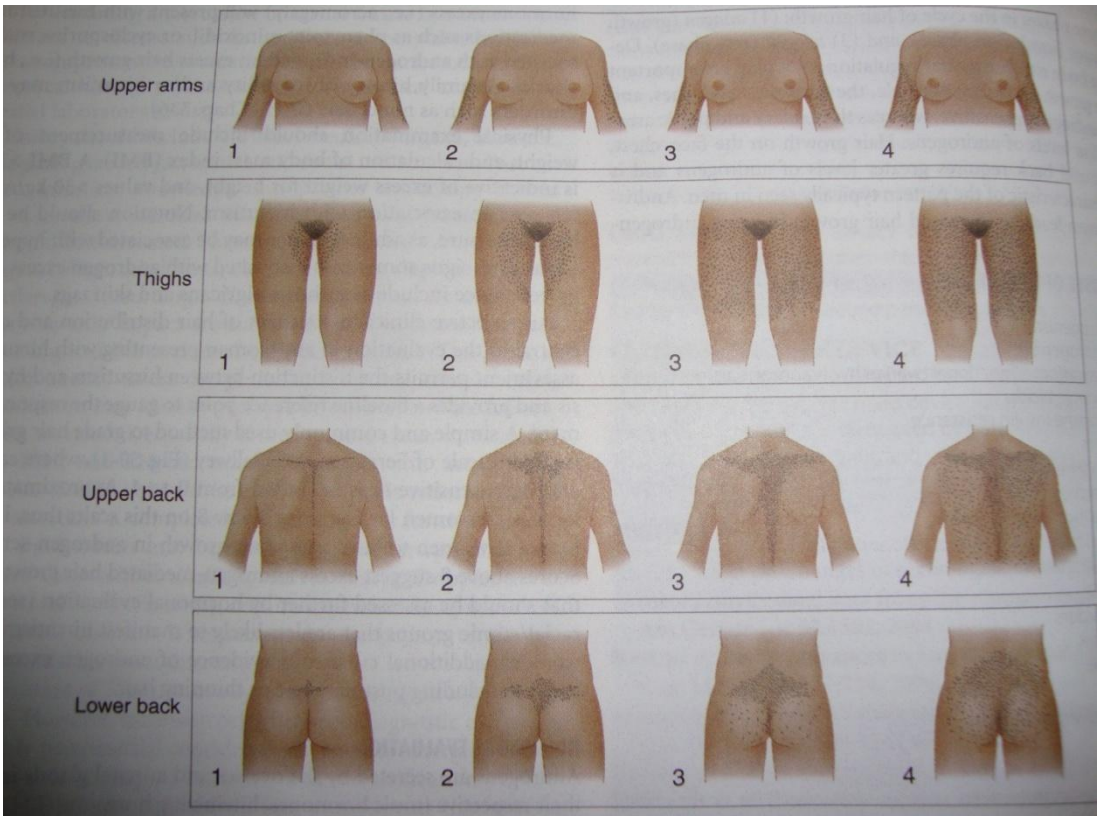
An objective clinical assessment of hair distribution and quantity is central to the evaluation in any woman presenting with hirsutism. This assessment provides the baseline reference point to gauge the response to

treatment. A simple and commonly used method to grade hair growth is classical or modified Ferriman- Gallway scale where each of nine androgen sensitive sites is graded from 0 to 4.[29] It is normal for most women to have some hair growth in androgen sensitive areas. Score of 8 or more suggest excess androgen mediated growths.[30]

	1	2	3	4
Upperlip	Small number of terminal hairs over outer lip border	Thin moustache covering less than 50%	Moustache covering 50% from margin or 50% the lip height	Moustache covering most of upper lip & crossing the midline
Chin	Sparse terminal hair on chin	Sparse terminal hair with small thickened areas	Entire chin covered with light growth	Entire chin covered with heavy growth
Chest	Circumareolar or midline terminal hair	Circumareolar and midline terminal hair	75% of chest covered with terminal hairs	Entire area covered with terminal hair
Abdomen	Scattered midline terminal hairs	More terminal hairs, still midline	50% of upper abdomen covered	Entire area covered with terminal hair
Pelvis	Small number of scattered midline terminal hairs along the length of linea alba	Midline concentration of terminal hair along the length of linea alba	A midline thickened band of terminal hair less than ½ width of pubic hair	An inverted V shaped coverage ½ width of pubic at base

Upper arms	Scattered terminal hair over less than 25% of upper arm	Increased but incomplete coverage	Entire area covered with light growth	Entire area covered with heavy growth
Thighs	Scattered terminal hair over less than 25% of thigh	Increased but incomplete coverage	Entire area covered with light growth	Entire area covered with heavy growth
Upper back	Sparse terminal hairs over upper back	Increased number of spread terminal hairs	Entire area covered with light growth	Entire area covered with heavy growth
Lower back	Sacral area with hair coverage less than 4 cm wide	Increased sides coverage	75% of lower back covered with terminal hairs	Entire area covered with heavy growth





Other methods for measuring hirsutism:[24]

Hatch and colleagues method: This is also a variation of the Ferriman Gallwey technique. The lower legs and lower arms were not included.

Lorenzo' s study of hirsutism: Its score pattern was based on only five body regions namely chin, upper lip, chest, abdomen and thighs. Abraham' s classification based on Ferriman Gallwey score:[12]

Score	**Presentation**
<8	Normal
8 – 16	Discrete hirsutism
17 – 25	Moderate hirsutism
>25	Significant hirsutism

Hormonal evaluation:[31]

Total testosterone: Early morning plasma total testosterone estimation is usually done on days 4 to 10 of the menstrual cycle.[16] If it is more than 200ng/ml, more extensive investigation for virilising tumour to be done.[32]

- Free testosterone: The fraction of testosterone that is not bound to its carrier protein, sex hormone binding globulin, is biologically available for conversion to DHT and for binding to androgen receptors.

- Sex hormone binding globulin (SHBG): Hyperinsulinemia and/or androgen excess decreases hepatic production of SHBG, resulting in elevated levels of unbound testosterone.

- Dehydroepiandrosterone sulphate (DHEAS): It is a supplementary test if there is raised testosterone. More than 7 microgram/ml suggests an adrenal tumour.

- LH to FSH ratio: Increased ratio (>2) is suggestive of PCOS.[17]

- Serum FSH and estradiol levels: Measured in women presenting with oligo/anovulation to exclude the possibility of hypogonadotropic hypogonadism (ovarian dysfunction of pituitary origin).

- 17 hydroxy progesterone: Measurement of serum 17-hydroxyprogesterone is useful to differentiate between late onset

congenital adrenal hyperplasia and PCOS. If the early morning 17 hydroxyprogesterone is <200ng/dl or if ACTH stimulated 17 hydroxy progesterone level is <1000ng/dl, late onset CAH is unlikely to be the diagnosis.

- Serum prolactin: Evaluated for hyperprolactinemia, but also elevated in PCOS, acromegaly and thyroid disorders.

- 24-hour urine cortisol:

Urine cortisol values of greater than 50 to 100 μg/dl in an adult suggest the diagnosis of Cushing's syndrome. This is considered as the most specific test.

- Overnight low-dose dexamethasone suppression test:

1 mg of dexamethasone is given at 11 PM, plasma cortisol level at 8 AM the next morning should be measured. In unaffected individuals, usually the levels will be < 1.8 μg/dl. Because adrenal androgens are readily suppressed by low doses glucocorticoids, the dexamethasone androgen suppression test distinguishes ovarian from adrenal androgen overproduction.

- Thyroid function test: TSH, free T3. Hirsutism has been reported with congenital hypothyroidism.[33]

- GnRH analogue & ACTH combined stimulation test:

100 microgram of GnRH analogue is given subcutaneously. Patients with subtle hyperandrogenemia are expected to show higher response of 17 hydroxyprogesterone and total testosterone. ACTH 250 microgram is also administered as intravenous infusion. Again, patients with occult hyperandrogenemia show increased responses of 17 hydroxy progesterone, total testosterone and plasma cortisol.

- Somatomedin-C level: Screened for acromegaly.
- Glucose tolerance test
- Lipid profile
- USG abdomen: Useful to identify ovarian mass
- CT/MRI: Used to localise adrenal mass or pituitary tumour

Serological indices in hirsutism:

Although the testosterone level does not correlate with the severity of hair growth, it correlates with the complex formula given below:[34,35]

(Testosterone/SHBG) + (Androstenedione/100) + (Dehydroepiandrosterone sulfate/100)

Index of free testosterone = (100×Total Testosterone)/SHBG

Medical treatment: [36]

Oral contraceptives

Antiandrogens

- Cyproterone acetate
- Drospirenone
- Spironolactone
- Flutamide

Enzyme inhibitors

- Finasteride
- Eflornithine

Insulin-sensitizing agents

- Metformin
- Rosiglitazone
- Pioglitazone

Gonadotropin releasing hormone analogues

- Leuprolide
- Nafarelin

Adjuvants:

Epilation methods

- Physical and chemical epilation
- Electrolysis or electroepilation
- Laser/photoepilation

The medical treatment of hirsutism is used to rectify any causal hormonal imbalance and improve the aesthetic appearance of hirsutism. The primary goal is to achieve central or peripheral androgen suppression using four groups of drugs like androgen production inhibitors (oral contraceptives, gonadotropin releasing hormone [GnRH] analogues), peripheral androgen blockers (cyproterone acetate, flutamide, and spironolactone), enzyme inhibitors (finasteride), and insulin-sensitizing agents (rosiglitazone, metformin).

Oralcontraceptives:

Combined oral contraceptives containing ethinyl estradiol 20- 35 microgram and one of the newer progestogens (desogesterol & gestodene) or cyproterone acetate are the most suitable for the treatment of androgenic symptoms because of their relative non androgenicity. This less androgenic effect induces a significant increase in SHBG and IGF binding protein with a resulting reduction of free testosterone.[37]

Antiandrogens

Cyproterone acetate:

Cyproterone acetate (CPA) acts mainly by blocking androgen receptors in target organs, and also reduces the activity of skin 5α-reductase, the enzyme responsible for converting testosterone to the more

potent androgen, dihydrotestosterone. It also suppresses gonadotropin secretion thereby reducing ovarian and adrenal androgen production.[38]

The progestin activity of CPA requires the drug to be combined with estrogens in women. Patients not desiring conception can be given cyclic cyproterone acetate 2mg/day along with ethinyl estradiol 35 microgram/day for 21 days followed by a 7 days rest. No clinical differences in outcome were found between cyproterone acetate and the other medical therapies like ketoconazole, spironolactone, flutamide, finasteride, and GnRH analogues.[39] Cyproterone acetate can lead to liver function abnormalities, weight gain, loss of libido and menstrual irregularity.

Spironolactone:

Spironolactone is an aldosterone antagonist and androgen receptor antagonist, exhibits a dose-dependent competitive inhibition of the androgen receptor. It also inhibits 5α reductase enzyme.[40] Spironolactone is an effective treatment for androgen-dependant hirsutism in doses ranging from 50 to 200 mg/day. Spironolactone in combination with OCPs is more effective than OCPs alone.[38]

Spironolactone can elevate potassium by blocking the effect of aldosterone on the kidney. Blood pressure and potassium concentrations should be screened every 4 weeks. Spironolactone should not be used to treat hirsutism in women with renal insufficiency or hyperkalemia.

Drospirenone:

Drospirenone, a progestin used in several OCPs, is also an antiandrogen. Drospirenone 3 mg (the dose used in OCPs) is roughly equivalent to spironolactone 25 mg or CPA 1 mg. It improves the clinical signs of hirsutism via antiandrogenic and antimineralocorticoid action.[41] There is a significant fall in serum total and free testosterone levels with a concomitant increase of sex-hormone-binding globulin (SHBG).[42]

Flutamide and bicalutamide:

Flutamide is a nonsteroidal compound that seems to act only at the androgen receptor site and is therefore considered a pure antiandrogen agent. The serum half-life of flutamide is approximately 6 hours. Flutamide is usually used in dosages ranging from 250 to 750 mg daily. Liver toxicity is a rare but potentially severe side effect. Serum transaminases should be measured frequently. Some side effects such as dry skin, diarrhoea, nausea, and vomiting have also been reported.

Low-dose bicalutamide 25 mg/day is a new potent orally active well-tolerated, non steroidal pure antiandrogen with a halflife of 7–10 days. Bicalutamide has been shown to be an effective drug in the treatment of patients with hirsutism due to PCOS and also in idiopathic hirsutism with no significant side effects or modifications in menstrual cycles. Hepatotoxic effects have been reported at doses of 50 mg per day.

Enzyme inhibitors

Finasteride:

Finasteride is an antiandrogen which competitively inhibits type 2 isoenzyme of 5α –reductase.[16] Thereby it decreases the amount of hormone available peripherally for interaction with the androgen receptor without altering ovarian and adrenal androgen secretion. The serum halflife of finasteride is 6 to 8 hours. Interestingly a single dose of finasteride suppresses serum DHT levels for up to 4 days. This phenomenon is probably due to the high affinity of finasteride towards 5α reductase enzyme.

Finasteride is used in doses of 1 mg to 5 mg/day. It can lower hirsutism scores by 30–60% in addition to reducing the average hair diameter.[43] The use of any drug with antiandrogen effect requires a safe and effective method of contraception in order to avoid the potential risk

of feminization of a male fetus. Considering the halflife of all mentioned drugs used in the treatment of hirsutism, it seems to be safe for a woman to become pregnant after discontinuation of the therapy for 10 days.

Insulin-sensitizing agents

Rosiglitazone and metformin:

Insulin sensitizing agents are metformin, rosiglitazone, and pioglitazone. They are widely available standard medications for the treatment of non insulin-dependent diabetes mellitus. When given to non diabetic subjects, they lower only insulin levels, not blood sugar levels.

Metformin decreases hepatic glucose production, lowers insulin levels with improved insulin sensitivity and has an elimination halflife of about 6 hours. Thiazolidinediones (TZDs) improve the action of insulin in the liver, skeletal muscle, and adipose tissue. By reducing hyperinsulinemia, both metformin and TZDs reduce adrenal & ovarian androgen biosynthesis and raise levels of SHBG[44,45] Thereby they reduce the circulating free & biologically active androgens and ameliorate hirsutism. In vitro studies demonstrated a direct effect of metformin on ovarian steroidogenesis.[46] When using rosiglitazone and pioglitazone, a regular monitoring of liver enzyme levels is recommended.[44]

Although metformin seems to be safe during pregnancy with no evidence of teratogenicity, it should be discontinued during pregnancy as there are no sufficient epidemiological data. Rosiglitazone and pioglitazone are Class C drugs with evidence of teratogenicity in animals. They should not be used in women of childbearing age unless they are strictly combined with an effective method of contraception.

Gonadotropin–releasing hormone (GnRH) analogues:

Leuprolide acetate, a GnRH agonist causes sudden release of gonadotropins that result in desensitization and downregulation of GnRH receptors. This leads to suppression of testosterone and estradiol synthesis. It is used as a rescue medication in patients not responding or not tolerating OCPs. It is administered as a depot preparation of 7.5mg intramuscularly monthly along with 25 -50µg transdermal estradiol.[7] Side effects include hot flushes, osteoporosis and atrophic vaginitis.

Hair removal methods:

Hair removal methods include shaving, waxing, sugaring, threading, use of tweezers, depilatory creams, electrolysis (so called electroepilation) and laser epilation. The choice of epilation technique

depends largely on personal preferences, region to epilate, colour of hair and cost.

Physical and chemical methods: [4,36]

Shaving is considered as being rapid, effective, simple, and cheap. The only disadvantage is that shaving has to be repeated daily and folliculitis frequently develops when hair regrows.

Electric rotating epilating devices or waxing techniques (cold or warm waxes) are both based on hair shaft epilation leading to longer lasting hair-free effects. The procedure must be repeated every 2–6 weeks. The disavantages are pain, inefficient epilation of short hair, skin irritation and folliculitis.

Sugaring is a variant of epilation where a mixture of lemon acid 8.0, sugar 65.0, glucose 15.0, and aqua purificata 100.0 is used.[36] After being intensely mixed, the paste is applied for 0.5 cm thickness on the area to be epilated and taken off with a rapid movement.

For chemical depilation the hair is dissolved by separating the disulfide bonds and the peptides of the hair keratin. The depilatory creams used today are mostly based on thioglycolates 2–4%. Calcium salts of the thioglycolates are preferred as they have very low irritating

effect in form of creams, lotions, or aerosol foams. After a 5 to 15 minute leave-on time, they are taken off together with the dissolved hair shafts. Disadvantage of this method is the irritating potential as, not only the hair keratin but also upper layers of the epidermis is affected. This method is preferably used on the legs and should be avoided in skin folds.

Bleaching of hair helps to make excessive hair less visible. For home use, 12% hydrogen peroxide preparations as aqueous solutions are used, which are mixed just before usage together with ammonia-based solutions or creams and applied for 30 minutes. For any bleaching or chemical depilation, a small test area should be done first. Disadvantages of bleaching are irritation and sensitization potential of peroxide/sulfates which are part of most commercial products. Bleaching is suited for upper lip, chin, and arms.

Eflornithine:

Eflornithine is the first topical drug for the treatment of unwanted facial and chin hair. It slows hair growth. The cells in the hair follicle undergo rapid growth and maturation as they transform into hairs. Polyamines are needed for this rapid cell growth and differentiation. The production of these polyamines depends on the activity of an enzyme, ornithine decarboxylase (ODC). Eflornithine is an irreversible inhibitor of

ornithine decarboxylase [32] and thereby slow the growth and differentiation of the cells within the hair follicles. It delays the initiation of anagen and keeps hair in telogen.

A topical preparation, Eflornithine hydrochloride cream 13.9% is approved for the treatment of unwanted facial hair in women. Systemic absorption is extremely low. The common side effects seen with eflornithine are acne, erythema, pseudofolliculitis barbae, headache , skin itching, burning or tingling, rash, and ingrown hairs.[6] Less common side effects are swollen lips, nausea, numbness, contact dermatitis, and herpes simplex.[47] Eflornithine was approved by the FDA in July 2000. Facial hair thinning occurs usually after two months.[48]

Electrolysis – electroepilation:

Electrolysis is a technique that can lead to permanent destruction of the hair follicles. An electric current is delivered by a probe placed in contact with the hair. An epilation probe is directly introduced in the hair follicle opening. It damages the hair follicle either by direct current (galvanic electrolysis) or by high frequency alternating current (thermolysis); the latter destroys the hair follicle by heat. The “Blend method” combines both types of these currents and is considered as the most effective technique for electrolysis.

Per session about 100 hair follicles can be treated**.** Potential disadvantages are pain, follicular hyperpigmentation, scarring and sometimes infection. The best results were obtained when electroepilation was combined with medical treatment to resolve any androgen excess. Although permanent hair removal can be achieved in some cases, the success of the technique ultimately relies on the skill of the operator.[49]

Laser hair removal:

Lasers cause permanent hair reduction rather than permanent hair removal as wrongly claimed.[50] It is defined as a noticeable reduction in the number of terminal hairs. Laser treatment usually produces complete but temporary hair loss for 1-3 months followed by partitial but permanent hair loss. There is noticeable reduction in the number of growing hair. Also, regrowing hairs are thinner and lighter in colour due to miniaturization of hair follicles.[51]

History:

Goldman and colleagues first described the injury of pigmented hair follicles by ruby laser in 1963. Subsequently Oshiro and Maruyama noted hair loss after treatment of naevi with ruby lasers. The possibility of selectively destroying hair follicles without causing extensive epidermal

damage was suggested with the introduction of the concept of selective photothermolysis (Anderson and Parrish 1983). Grossman and colleagues reported the first clinical controlled study of laser hair reduction in 1996 using ruby laser.

Hair biology: Considerations for laser hair removal:[52,53]

- For effective long term hair removal, laser should damage the main structures responsible for hair growth – bulb and the stem cells.
- Melanin, the key target chromophore is present in the hairbulb.
- Melanisation occurs only during the anagen phase and hence only hairs in anagen phase are susceptible.
- Darker the hair colour, more is the melanisation and hence larger is the quantum of target chromophore available for effective laser hair removal.
- Thicker the hair, larger is the hairbulb and follicle area. This results in longer thermal relaxation time requiring longer pulse duration.

LASER physics:[54,55]

Physical properties of laser light:

- Laser light is formed of monochromatic photons (light from a given source is of single wavelength).
- Laser light is coherent (all the photons from laser source are emitted with the same wavelength, travel in the same direction and in the same phase).
- Laser light is collimated (laser beam has a constant cross sectional diameter).
- Laser light has high intensity which is required for transmission of maximum energy.
- Laser light can be delivered in pulse mode.
- Spot size of the laser beam determines its depth of penetration.

Parameters of laser light:

The interaction of laser energy with the tissue depends on a number of factors including power, spot size, duration of exposure, wavelength and tissue properties.

- Energy that is contained in the light is expressed in joules.
- Fluence or energy density is the actual amount of energy applied to the unit area of target tissue. It is expressed as joules/cm^2.
- The wavelength of laser light gives its characteristic colour.
- Pulse duration is the amount of time laser energy is applied (ns,ms).
- Pulse frequency is measured in hertz. It is the repetition rate of pulse.
- The laser light is delivered in two broad categories: continuous wave or pulse wave.

In the continuous wave mode, there is an uninterrupted beam of radiation of relatively low power.

In the pulse mode, the continuous beam can be interrupted to form pulses. This is achieved by cutting the beam either with simple mechanical shutters, mode locking or by Quality switching. Superpulsed and ultrapulsed lasers produce higher peak power with lesser pulse durations.

Laser - Tissue interactions:[56]

A variety of interactions occur with exposure of the skin to laser light. The light may either be reflected back from the skin surface, scattered within the tissue, transmitted through the tissue, absorbed by the tissue or there can be combination of these effects. The absorption of laser energy results in its conversion to thermal energy. There are three means by which light can cause follicular damage: photothermal, photomechanical, or photochemical. Photothermal injury is the principal mechanism of action for most currently available lasers.

Selective photothermolysis:[57]

The tendency of chromophores to absorb energy is wavelength dependent.

The theory of selective photothermolysis postulates that light of a wavelength which is absorbed by a target chromophore will selectively damage or destroy that chromophore if the fluence is sufficiently high and the pulse duration is less than or equal to the thermal relaxation time of that chromophore. If the pulse duration is less than the thermal relaxation time, heat diffusion does not take place and damage is selectively confined to the target.

The concept of selective photothermolysis has been successfully used in laser hair removal. Melanin is the target chromophore in the hair for selective photothermolysis. The therapeutic window of wavelength for melanin is between 630 – 1100 nm. However, melanin in the epidermis presents a highly competent site for absoption. Selective cooling of the epidermis is found to minimize the epidermal damage.[58] Various cooling agents and systems such as ice pack, layer of cool gel, cooled glass chambers, cooled sapphire tip, pulsed cryogen spray and cold air are used to effectively cool the epidermis.

Available hair removal systems:[59]

Laser hair removal systems are grouped into three categories, depending on their wavelength and type of light source:

1. Red light lasers (Ruby laser - 694nm)

2. Infra red lasers (Alexandrite laser - 755nm; Diode laser - 800nm, Nd : YAG laser - 1064nm)

3. Intense – pulsed light (IPL) source (550-1200nm) with appropriate filters [60]

All systems are able to temporarily interrupt hair growth, however, permanent reduction of hair density is mainly based on the number of

sessions, fluence and intensity of the hair color. Blond, red and white hairs are not suited for laser epilation, whereas dark hair on fair skin is the optimal target. Often, regrowing hairs are finer and lighter and a certain reduction is achieved for every session. Depending on the different published studies this can vary between 10–40%.

Q switched Nd YAG laser:

It delivers pulses in the nanosecond domain and is used for hair removal on all skin types. The Nd YAG laser has been proposed as an effective method of hair removal for several reasons. Firstly, its longer wavelength (1064 nm) allows for better skin penetration of the energy. Secondly, the Nd YAG laser with a wave length of 1064nm is relatively less absorbed by cutaneous chromophores. Finally it can treat a large amount of skin surface quickly.

The reduced melanin absorption at this wavelength necessitates the need for high fluences in order to adequately damage hair. However, the poor melanin absorption at this wavelength coupled with epidermal cooling device makes the Nd YAG laser a safer laser treatment for darker skin types up to phenotype VI.

Intense pulsed light:

Intense pulsed, non laser light sources, emitting non coherent, multi wavelength light have also been used for hair removal. By placing appropriate filters on the light source, wavelengths ranging from 590 to 1200 nm can be generated. Cut-off filters are used to eliminate short wavelengths, so that only the longer, more deeply penetrating wavelengths are emitted. Pulse durations vary in the millisecond domain. A single or multiple pulse (2-5), with various pulse delay intervals, can be chosen. The wide choice of wavelengths, pulse duration and delay intervals make the device potentially effective for a wide range of skin types.

Recently, IPL systems have been developed that are combined with 1064 nm laser light. These devices should allow for treatment of a wide spectrum of hair and skin colours.

Exogenous chromophore:

In persons with blonde, gray or white hair, endogenous chromophore, melanin, is deficient or absent and effective permanent laser removal cannot be achieved. For them rather than targeting endogenous melanin, an exogenous chromophore can be introduced into the hair follicle and then irradiated with light of a wavelength that

matches its absorption peak. The main problem is reliable penetration of the chromophore into all depths of the hair follicle. Therefore the technique in its present form is apparently inadequate for inducing permanent hair loss.

Contraindications:

- Superficial cuts/injuries
- Active infections
- Keloidal tendency
- Patients with unrealistic expectations

Side effects:

- Pain & discomfort
- Epidermal damage (vesiculation, crusting)
- Pigmentary alteration- de/hypo/hyper pigmentation
- Textural damage
- Local infection
- Scarring
- Allergy to topical anesthetic creams rarely, livedo reticularis, intense pruritus and urticaria.[61]

Patient selection:

- The best candidates for laser hair removal are individuals with black or brown hair.

- People with pale skin, with less melanin chromophore, are better candidates because the wavelength of laser energy used to destroy hair follicles is also readily absorbed by the epidermal melanin.

Pre procedure workup:

- A prior detailed history regarding presence of other medical problem (herpes labialis) or medications that contribute to the excessive hair growth, history of hypertrophic scars and keloids, history of any photosensitizing medication or isotretinoin intake within 1 year should be taken.

- Complete hormonal workup is necessary.

- 4-6 weeks prior to laser, sunscreens can be prescribed. Hydroquinone can be used in patients with dark skin.

- It is important to explain the patient that this is only hair reduction and not a permanent hair removal. The outcome and side effects must be explained at the same time.

- An informed consent is obtained. Photographs can be taken for records.

Procedure:

- Area to be treated is cleaned.
- Topical or local anesthesia is generally used on more sensitive areas.
- Depending on the equipment, cooling of the epidermis is achieved with a cooling gel applied before the delivery of a laser pulse.
- Ideal treatment parameters must be individualized for each patient.
- Hand piece is placed perpendicularly to the skin surface and is either held gently or pressed firmly depending on the laser systems.
- Laser pulses are placed adjacent to each other with minimal overlap.
- The normal immediate response is vapourisation of hair shaft followed, after a few minutes, by perifollicular edema and erythema.

Post procedure management:

- Ice packs are given to reduce postoperative pain / swelling.
- A topical antibiotic/steroid combination ointment is given.
- Patient is instructed to avoid sun exposure.

Schedule:

Repeated treatment is usually given when the hairs start to regrow (in general 4-6 weeks). Multiple sessions are required to achieve desired satisfactory results.

Advantages:

- Fewer sessions than electrolysis[62] / waxing.
- 50-60% permanency with prolonged hair removal.
- Less discomfort.
- Large areas can be treated in one session.
- Faster results.
- Fewer side effects.
- Better patient compliance.

Disadvantages:

- Expensive.
- Not effective for grey / white hair.
- Multiple treatment sessions

AIMS OF THE STUDY

- To know the prevalence of hirsutism among different age groups
- To identify the associated factors like acne, acanthosis nigricans, diabetes mellitus, hypertension and obesity
- To study the etiology of hirsutism
- To study the efficacy of Nd YAG laser & IPL in selected hirsutism patients
- To study the incidence of side effects following Nd YAG laser & IPL

MATERIALS AND METHODS

Study design	**:**	Prospective descriptive study
Study centre	**:**	Department of Dermatology, Rajiv Gandhi Government Hospital, Chennai
Study period	**:**	October 2009 to September 2011.
Sample size	**:**	Seventy three patients.
Inclusion criteria	**:**	All hirsutism patients with Ferriman Gallway score of 8 or more.
Exclusion criteria	**:**	Patients with Ferriman Gallway score of <8.

Clinical evaluation:

Complete history, general examination and dermatological examination were done for all patients included in the study by using a proforma. Investigations including postprandial blood glucose, lipid profile, USG abdomen & pelvis, hormonal assay - LH, FSH, testosterone, thyroid function test & prolactin were done for all patients. DHEAS & 17 hydroxy progesterone were done for indicated cases. With the investigation results, endocrine opinion was obtained for all patients. Polycystic ovarian disease was diagnosed based on Rotterdam Consesus Criteria.

Treatment protocol:

For all polycystic ovarian disease patients, Tab.Metformin 500mg half tablet at bed time was started by endocrinologist. The dose was gradually stepped up to half tablet twice a day or one tablet twice a day depending upon the regularity of the patient's menstrual cycle.

Patients who gave willingness for hair removal procedure were randomly allotted for Q switched Nd YAG laser and IPL. Those patients with keloidal tendency, systemic associations, grey hair and pregnancy were excluded. Duration of treatment, potential adverse effects and post treatment care were explained to the patients. Written consent was obtained. Treatment parameters in respect to pulse duration and fluence were determined according to manufacturer's recommendation. One cm square area grid on the treatment site was used to count hair at baseline and at subsequent intervals (0, 4, 8, 12, 20, 24 weeks). Hair over the treatment site was shaved on the day of procedure. Digital photographs were taken before each treatment session.

Nd YAG laser started from energy level 25%, frequency 2, increased in each sitting by 5%. Pulse duration was 20ns. Treatment was given for 6 sittings at a gap of 4 weeks.

For IPL laser, cooling gel was applied over the area before the procedure. Fluence started from 10 J/cm^2, was slowly increased by 5 J/cm^2 for each sitting. Pulse duration was maintained at 5 ms. Treatment was given for 6 sittings at an interval of 4 weeks.

The study was approved by the institute ethical committee. After the procedure, patients were advised to avoid sun exposure and physical sun screen was prescribed. Hair removal efficiency was calculated as a percentage of the reduction in the number of hairs at each visit compared with baseline count and was graded as mild (0 – 25%), moderate (26 – 50%), good (51 – 75%) and excellent (76 - 100%). All the patients were asked to grade their satisfaction level after the procedure as not satisfied, satisfied and very satisfied.

OBSERVATIONS

A total of 73 patients were included in the study.

Table 1 : Age distribution of the study population

Age group (years)	Frequency	Percentage
15 – 25	32	43.8
26 – 35	21	28.8
36 – 45	17	23.3
46 or more	3	4.1
Total	73	100.0

Most of the patients were in the age group of 15 to 35 years. The youngest patient was 15 years old and the oldest patient was 48 years old. Mean age of patients seen in our study population was 29.19 years.

Table 2 : Age of onset

Age group (years)	Frequency	Percentage
<15	19	26.1
16 – 20	19	26.1
21 – 25	17	23.3
26 – 30	11	15
31 – 35	5	6.8
36 – 40	2	2.7
Total	73	100

More than 50% of patients developed hirsutism before the age of 20 years.

Duration of the disease varied from 1 to 21 years.

Table 3 : Menstrual status of patients

H/o Menstrual irregularities	Frequency	Percentage
Yes	35	48%
No	38	52%

Menstrual irregularities due to various causes like PCOD, Cushing's disease, hypothyroidism and congenital adrenal hyperplasia were seen in 35 patients (48%).

Table 4 : Family history association

Family History	Frequency	Percentage
No	57	78.1
Yes	16	21.9

Around 22% of patients had positive family history of hirsutism.

Table 5 : Associations

Associations	Frequency	Percentage
Acne	13	17.8
Acanthosis nigricans	23	31.5
Androgenetic alopecia	11	15
Infertility	5	12.2(among married)

Most commonly associated cutaneous finding in our hirsutism patients was acanthosis nigricans (31.5%). Infertility was seen in 12.2% of married people.

Table 6 : Co-morbidities

	Frequency	Percentage
DM /HT	7	9.6
Lipid abnormalities	8	11

Recently hirsutism is considered as one of the markers of metabolic syndrome. So, all our patients were screened for diabetes mellitus, hypertension and lipid abnormalities. Features of metabolic syndrome were seen in 9.6% of patients. Among 7 patients with diabetes mellitus, 4 patients were above 40 years of age and 3 patients were below 40 years.

Table 7 : BMI association

BMI Group	Frequency	Percentage
Normal (18.5 – 24.9)	24	32.9
Excess wt. (25 – 29.9)	27	37.0
Obesity (30 – 39.9)	20	27.4
Morbid obesity (> 40)	2	2.7

Most of the patients in the study population (67%) were above the normal BMI range. Obesity was seen in 30% of patients. Mean BMI was 27.

Table 8: Etiological distribution

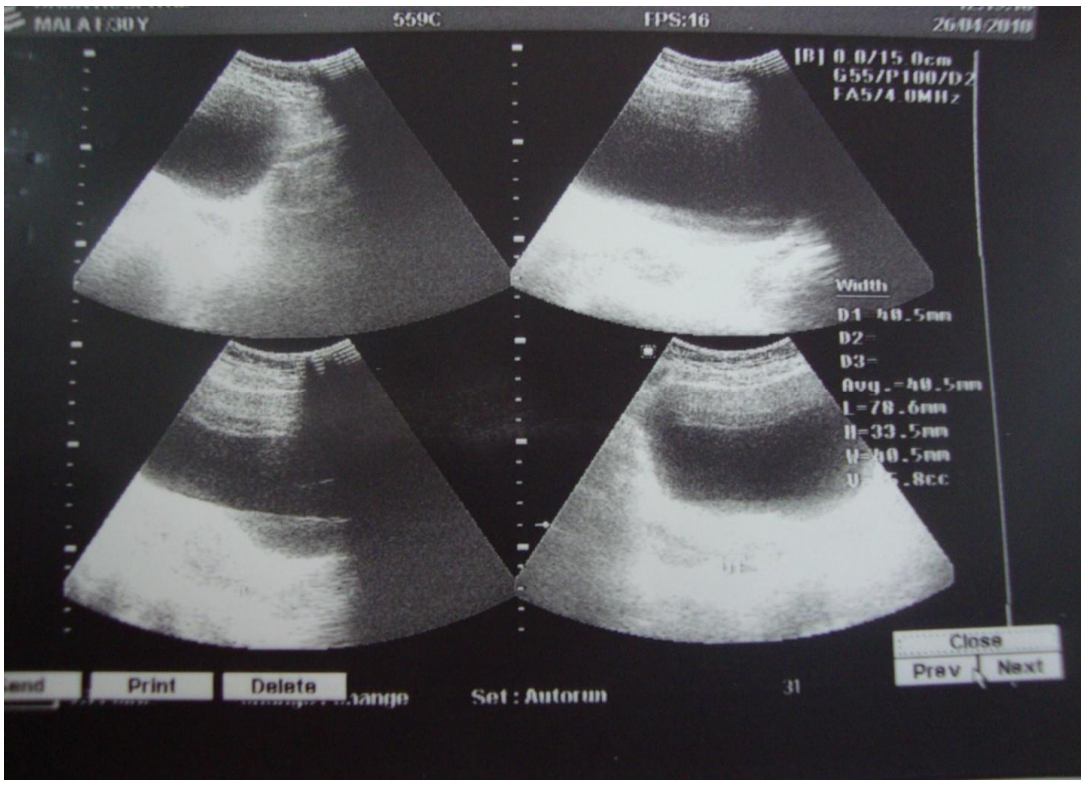
Etiology	Frequency	Percentage
PCOS	23	31.5
Hypothyroidism	7	9.6
Idiopathic	38	52.0
Drug	3	4.1
CAH	1	1.4
Cushing’s syndrome	1	1.4
Total	73	100

In our study, more than 50% patients were idiopathic. The next common cause was PCOS.

A patient with idiopathic hirsutism

A patient with polycystic ovarian disease

USG pelvis showing PCOS

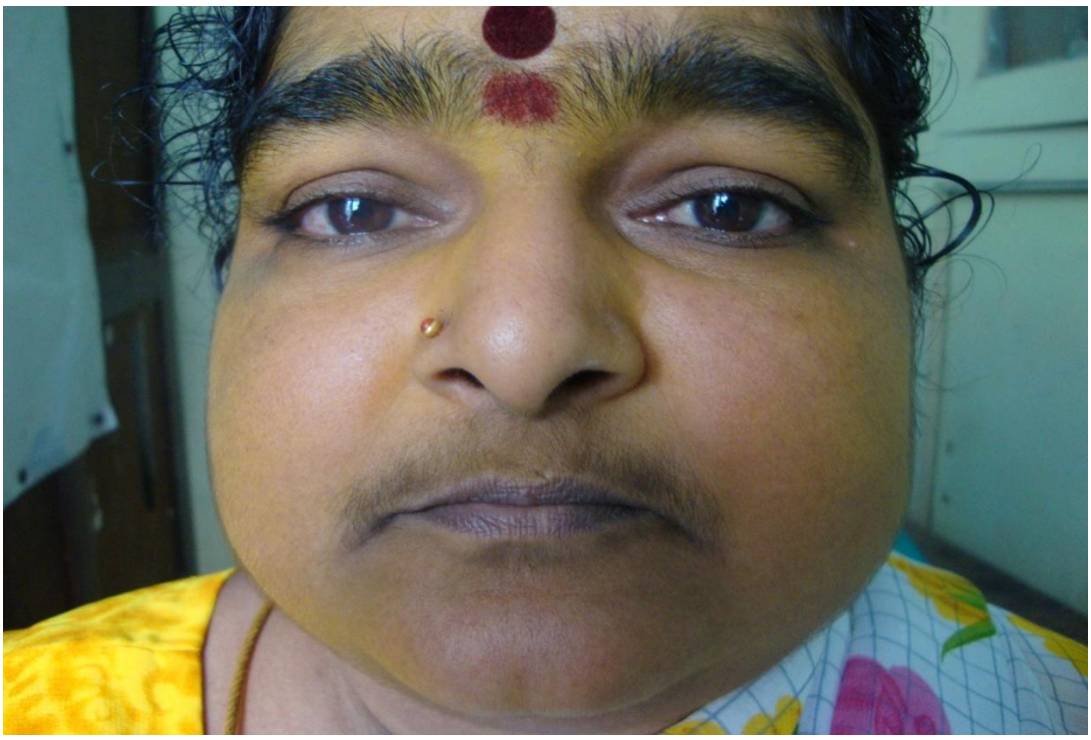


Hirsutism in a hypothyroid patient

Steroid induced hirsutism in a Pemphigus vulgaris patient

Cyclosporine induced hirsutism in a post renaltransplant patient

Hirsutism in cushing's syndrome



Hirsutism in a congenital adrenal hyperplasia patient

Table 9 : Ferriman Gallway Scoring

(Abraham's classification)

FG Score	Frequency	Percentage
Discrete (8 - 16)	53	72.6
Moderate (17 - 25)	20	27.4
Total	73	100.0

About 73% of patients had Ferriman Galway scoring (FG score) in the range of 8 to 16. The minimum score was 9 and the maximum score was 21. The maximum score was seen in a patient with congenital adrenal hyperplasia. Average FG score was 15.

Of the 73 patients, 40 patients were willing for hair removal procedure. They were randomly divided into two groups. The first group received Q switched Nd YAG laser. Second group was treated with IPL. We could not compare these two groups because of the small sample size and the unequal number of PCOS patients in the two groups.

Clinical evaluation:

Among the 23 cases of PCOS (diagnosed based on Rotterdam criteria), serum testosterone was elevated in 10 patients (43.4%). One PCOS patient had elevated prolactin level. LH, FSH ratio was more than 2 in 12 patients (52.1%). Polycystic ovaries were seen on pelvic

ultrasonogram in in 20 patients (86.9%) and DHEAS was elevated only in 5 patients (21.7%).

Among the total 73 patients. 7 patients had low T_4 level and high TSH level. One patient had elevated 17 hydroxyprogesterone (350ng). Plasma cortisol level was elevated in one patient which on dexamethasone suppression, became normal.

Q Switched Nd YAG Group:

Table 10 : Age distribution in Nd YAG Group

Age group	Frequency	Percentage
18 – 25	13	65.0
26 – 35	5	25.0
36 – 45	2	10.0

Large number of patients (65%) were below 25 years and were worried about their physical appearance.

Table 11 : Occupations of the study patients

Occupation	Frequency	Percentage
Working	5	25.0
House wife	4	20.0
Student	11	55.0

Majority of patients (55%) were students.

Table 12: Associated factors

	Frequency	Percentage
PCOS	6	30
Non PCOS	14	70

In the Q switched Nd YAG group, 6 patients had polycystic ovarian syndrome, all of them on T.Metformin. No other drug was given.

Table 13 : Skin types of study patients

Skin type	Frequency	Percentage
3	2	10.0
4	10	50.0
5	8	40.0

All the willing patients were randomly included in the study irrespective of their skin type.

Table 14: Areas of treatment in study patients

Site	No. of patients	Percentage
Upper lip	8	40.0
Upper lip & chin	5	25.0
Upper lip, chin & sides	3	15.0
Chin & sides	4	20.0

In this study, all the patients had hair growth over the face. Upper lip was the commonest site involved.

Table 15 : Hair removal efficiency (HRE) in Nd YAG group

Degree of improvement	No. of patients	Percentage
Mild (0-25%)	0	0
Moderate (26-50%)	2	10
Good (51-75%)	14	70
Excellent (75-100%)	4	20

In the Q switched Nd YAG group, 70% of study patients had a good response, 20% of patients had an excellent response and 10% of patients showed only moderate response.

Table 16 : Treatment sessions required for good response in NdYAG group

No. of sessions	No. of patients	Percentage
4	3	16.7
5	12	66.6
6	3	16.7

In all our patients, maximum hair removal was seen after 5 sittings. This is statistically significant. Energy level used in the 5^{th} sitting was 45%.

Table 17: Oneway ANOVA to find the maximum improvement between sittings in NdYAG Group.

Sittings	No. of patients	Mean HRE	S.D	P value
3	20	44.60	11.596	<0.001
4	20	56.10	7.847	
5	20	64.30	8.467	
6	20	65.05	8.787	

Table 18: Mean HRE in different skin types of the Nd YAG group.

Skin type	No. of patients	Mean HRE	S.D	P value
3	2	72%	8.485	0.392
4	10	65.7%	8.944	
5	8	62.5%	8.635	

Hair removal efficiency of Nd YAG laser was almost equal in the different skin types of our study patients.

Table 19: Independent samples t-test to compare the mean improvement between PCOS and non PCOS patients in NdYAG group.

	N	Mean HRE	S.D	P value
Without PCOS	14	64.14	9.256	0.496
With PCOS	6	67.16	7.935	

There was no difference in hair removal efficacy between PCOS and non PCOS patients.

Table 20 : Patient satisfaction for hair removal

Satisfaction levels	No. of patients	Percentage
Not satisfied	2	10
Satisfied	8	40
Very satisfied	10	50

Half of the study patients were very much satisfied with Nd YAG Laser. They felt that their facial appearance improved.

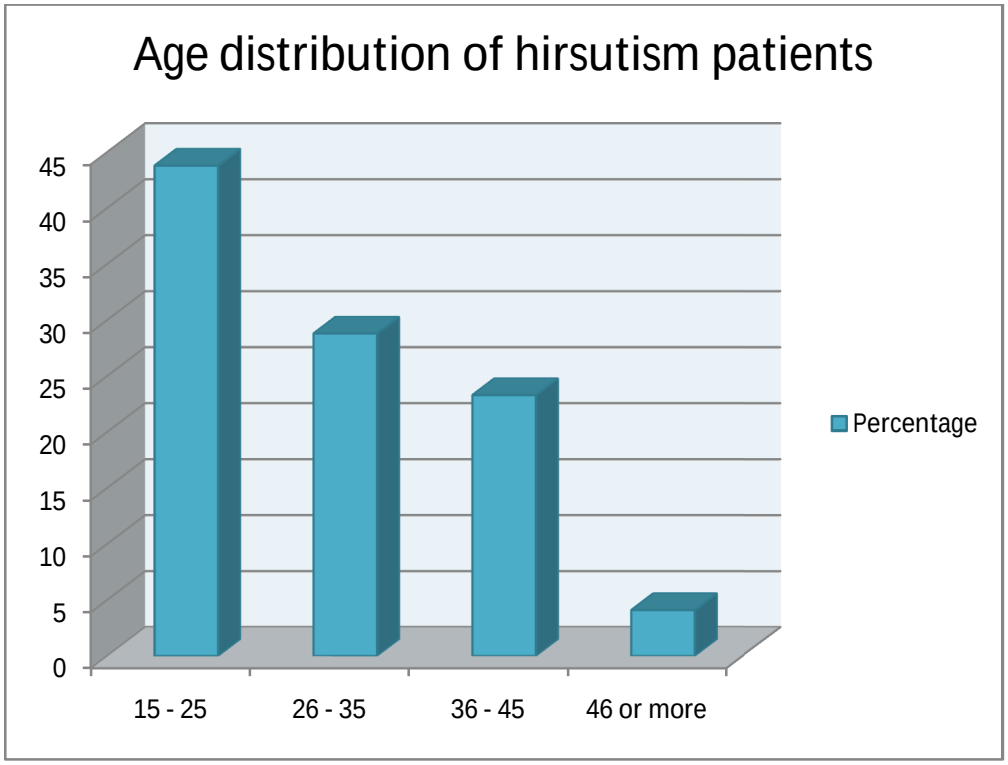
Table 21: Side effects seen in Nd YAG group

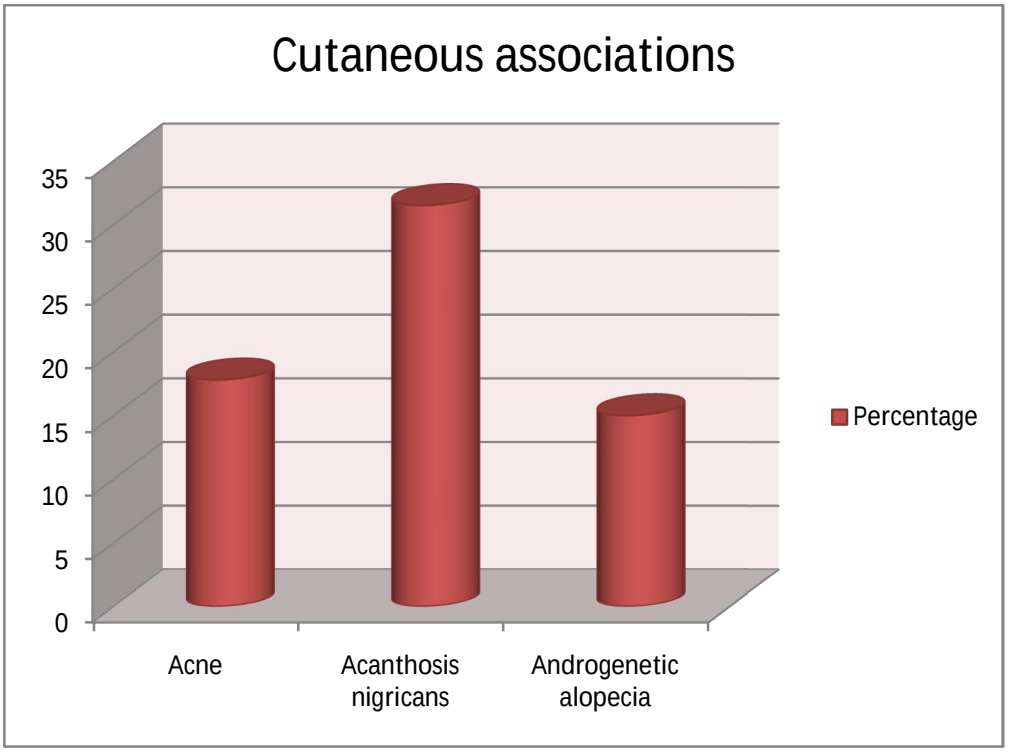
Side effect	No. of patients	Percentage
Transient erythema	18	90%
Perifollicular edema	18	90%
Pain	16	80%
Crusting	2	10%
Pigmentary disturbances	0	0
Scarring	0	0

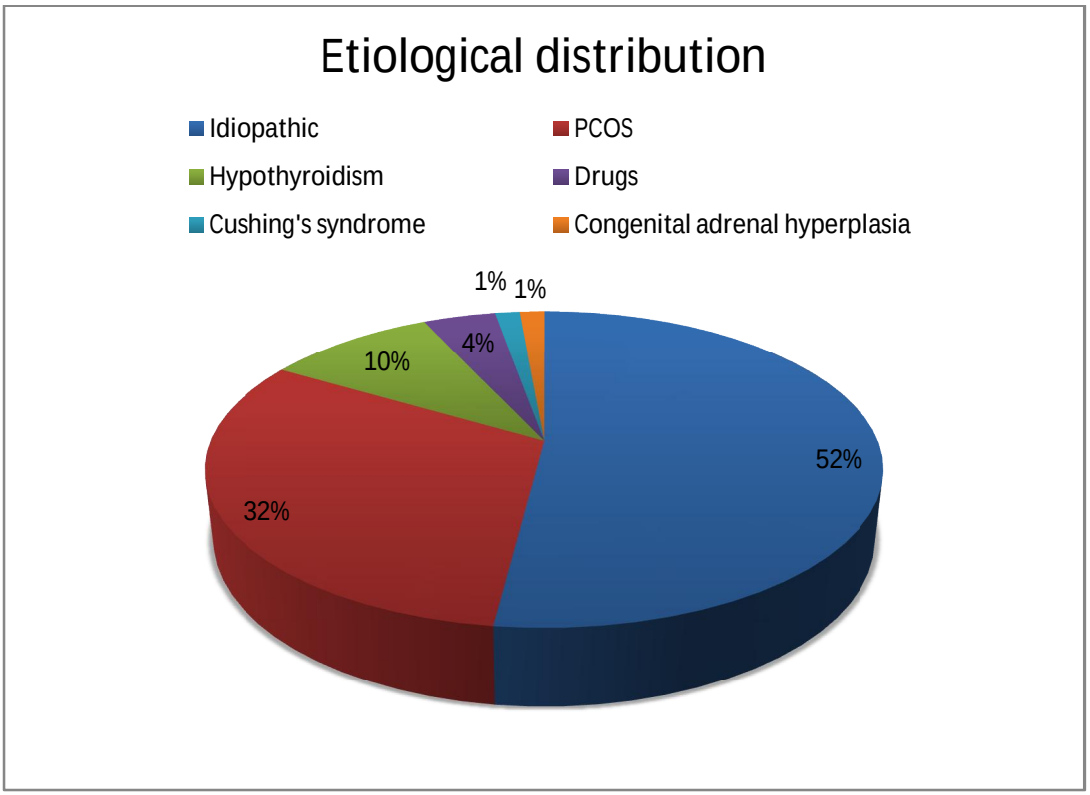
In the Q switched Nd YAG group, 90% of patients showed erythema and edema immediately after laser procedure which resolved spontaneously in 4-6 hrs. Crusting was seen in 10% of patients in the post procedure period.

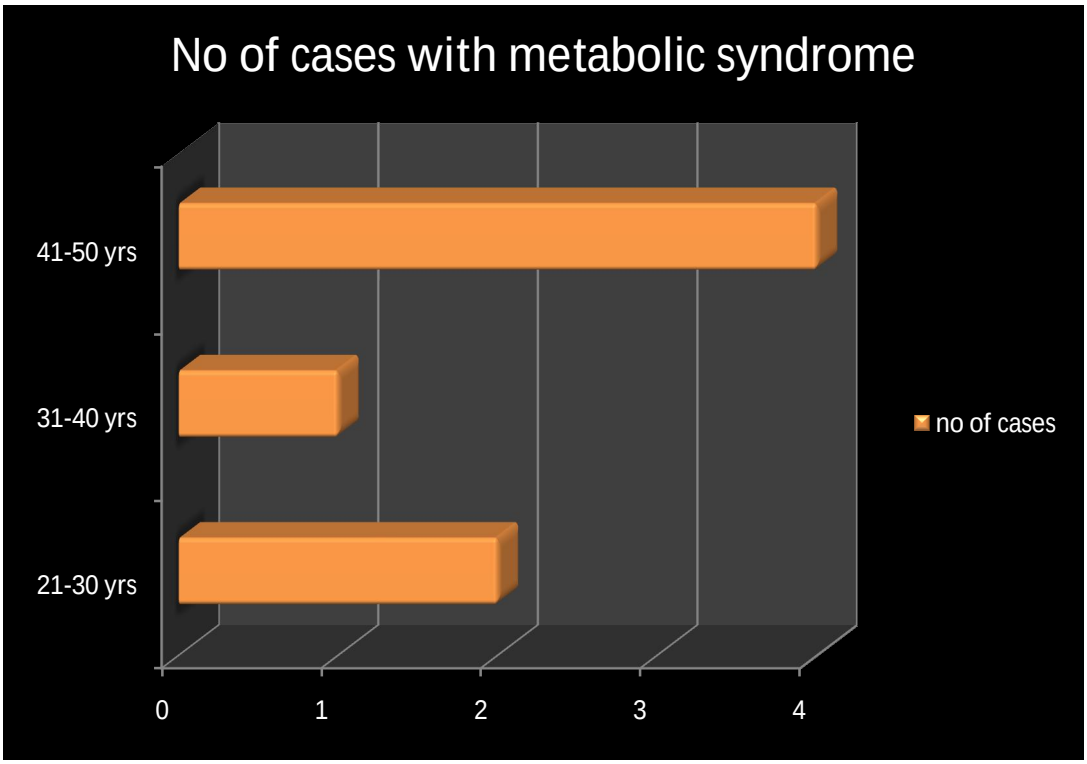
Follow-up:

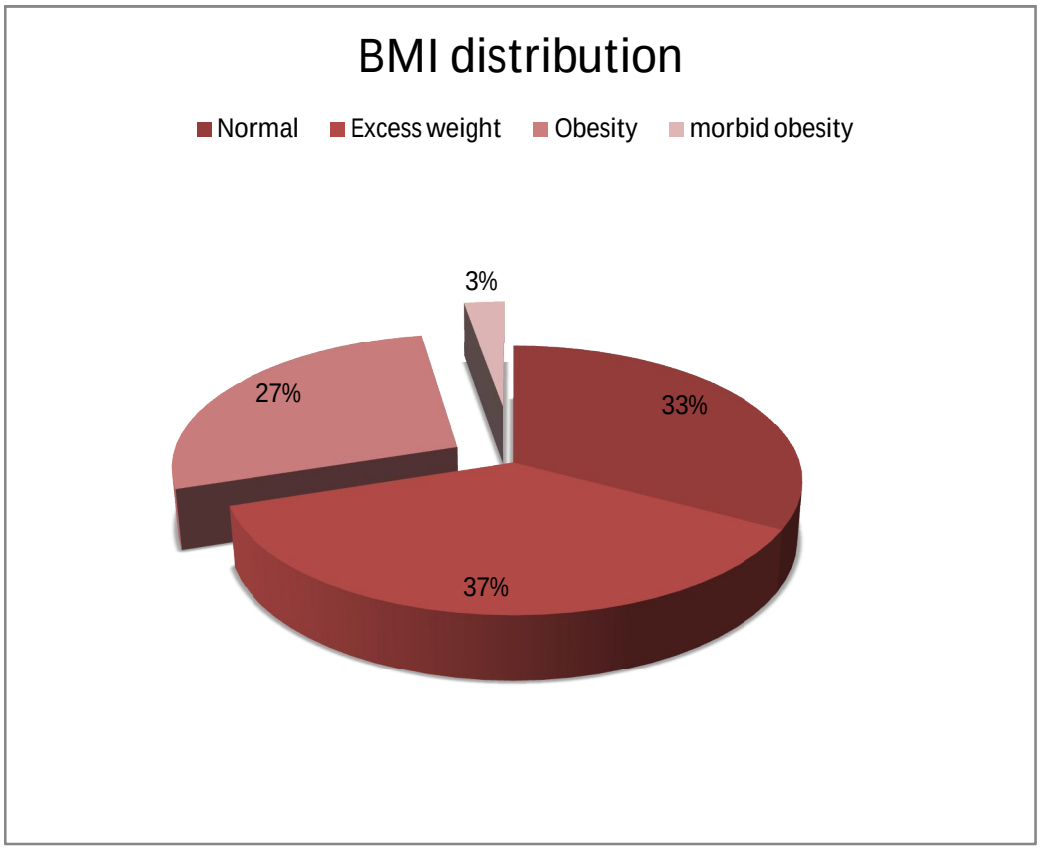
After 6 months, 10 patients came for follow up. Among them, one patient had hair regrowth of 30 - 40% and 9 patients maintained their results.

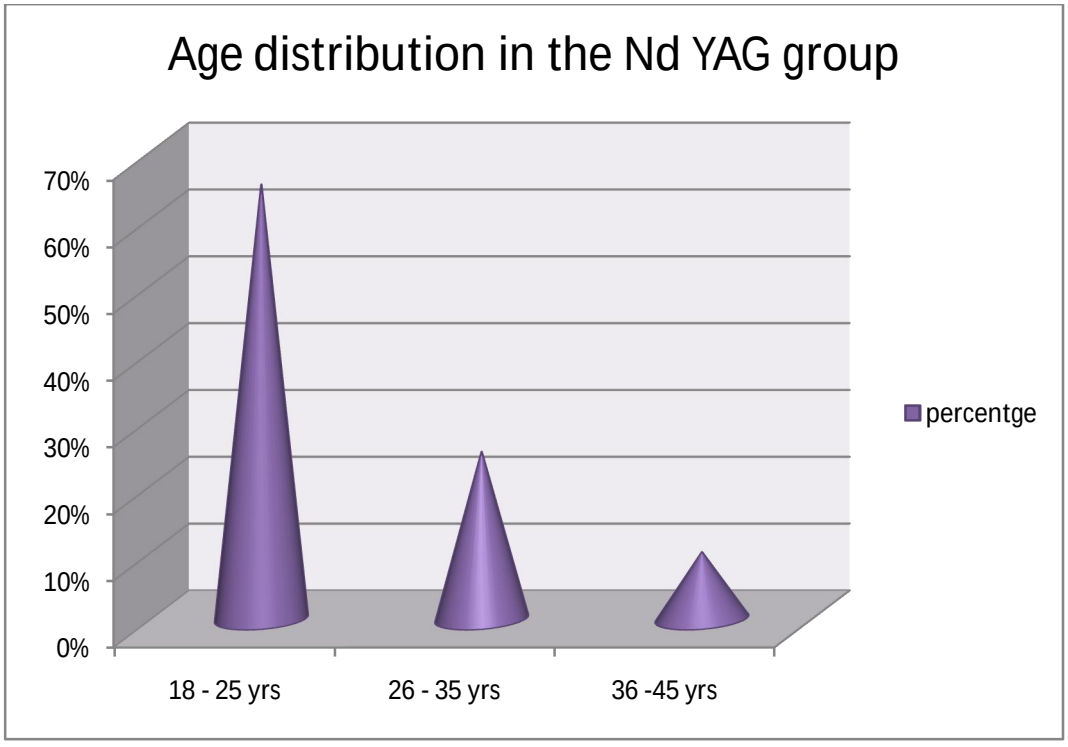

Age distribution of hirsutism patients
45
40
35
30
25
20
15
10
5
0
15 - 25
26 - 35
36 - 45
46 or more
Percentage

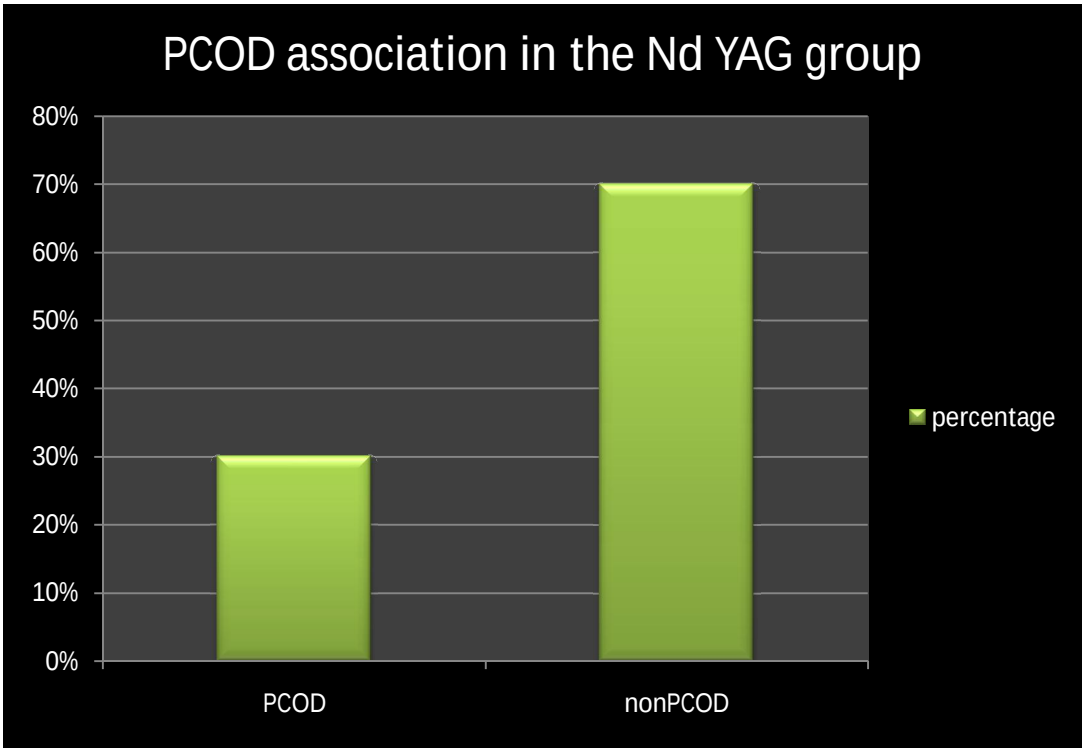

Cutaneous associations
35
30
25
20
15
10
5
0
Acne
Acanthosis nigricans
Androgenetic alopecia
Percentage

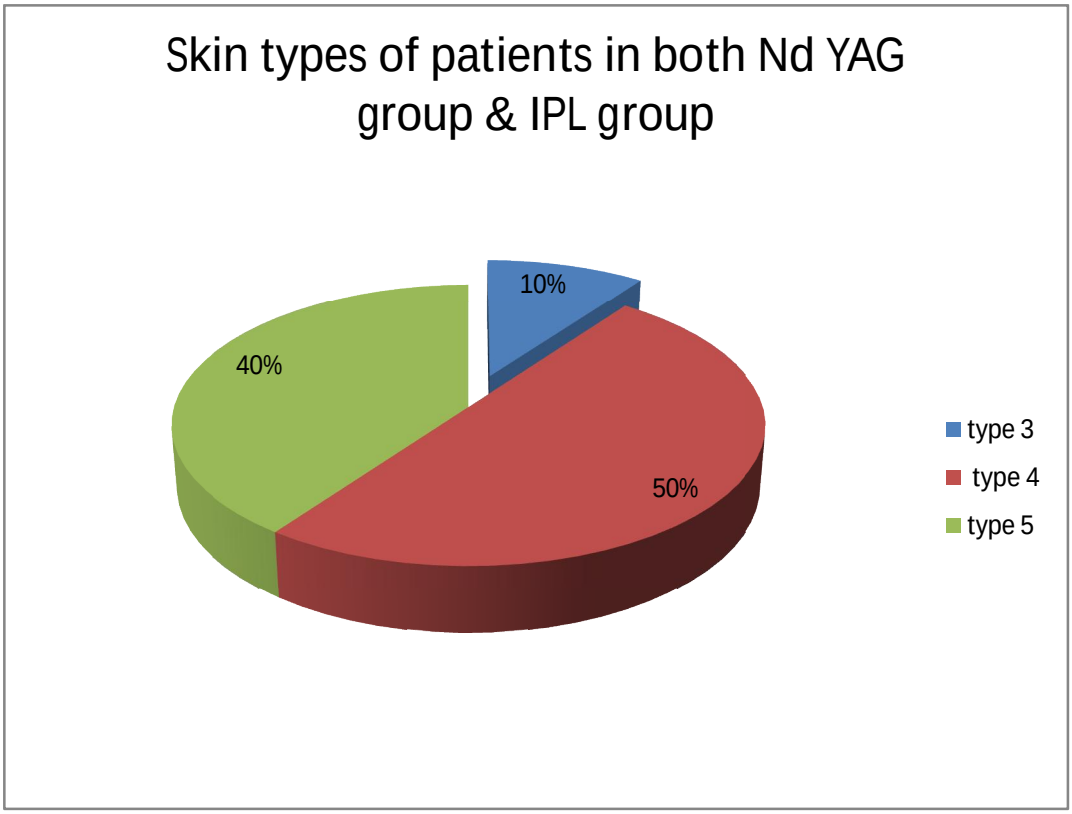

Etiological distribution
Idiopathic
PCOS
Hypothyroidism
Drugs
Cushing's syndrome
Congenital adrenal hyperplasia
1%
1%
4%
10%
52%
32%

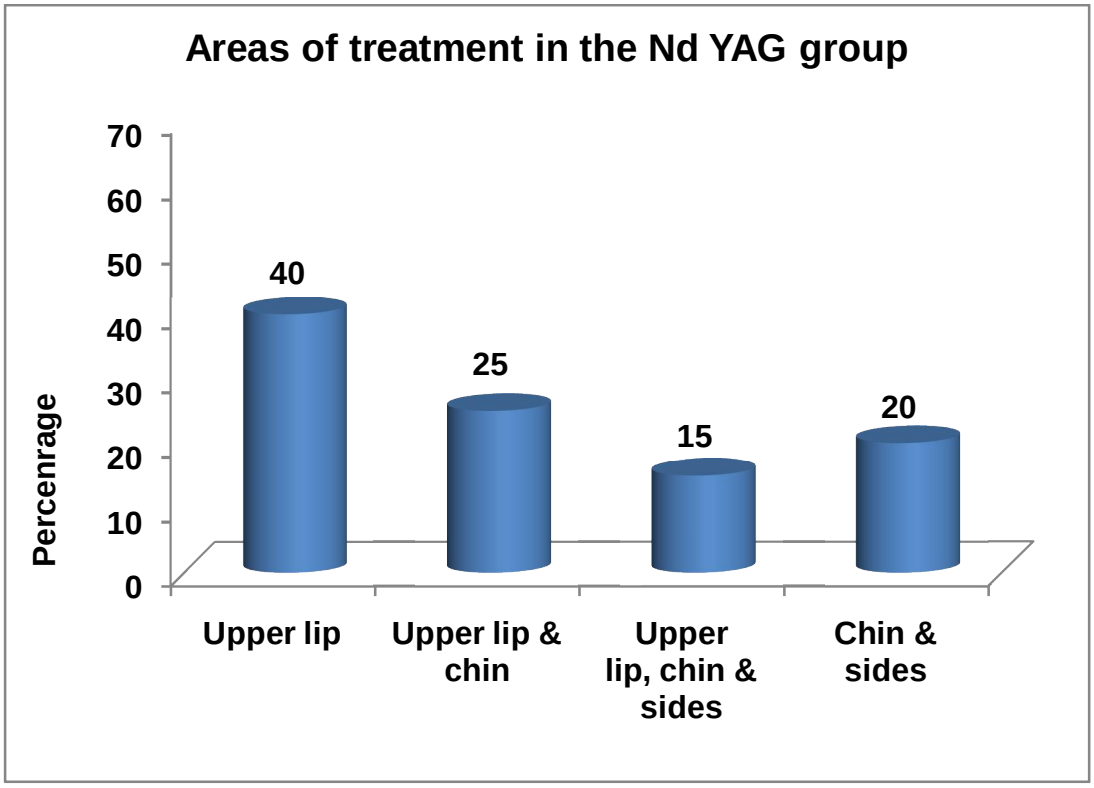

No of cases with metabolic syndrome
41-50 yrs
31-40 yrs
21-30 yrs
0
1
2
3
4
no of cases

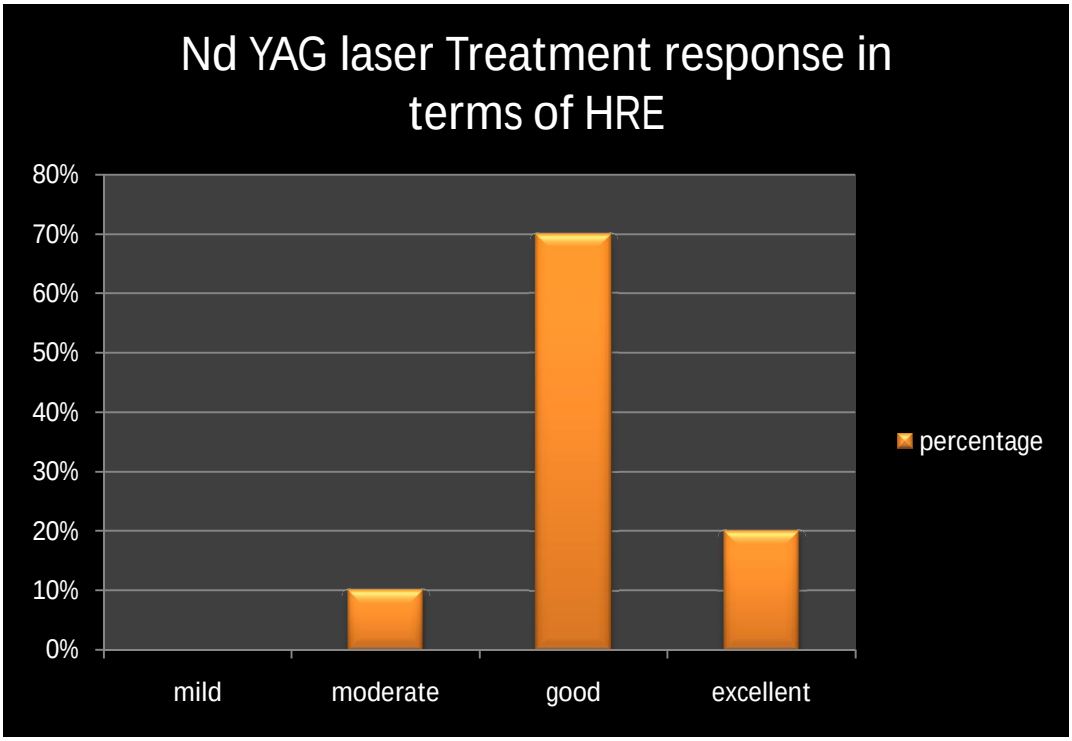

BMI distribution
Normal
Excess weight
Obesity
morbid obesity
3%
27%
33%
37%


Age distribution in the Nd YAG group
70%
60%
50%
40%
30%
20%
10%
0%
18 - 25 yrs
26 - 35 yrs
36 -45 yrs
percentge

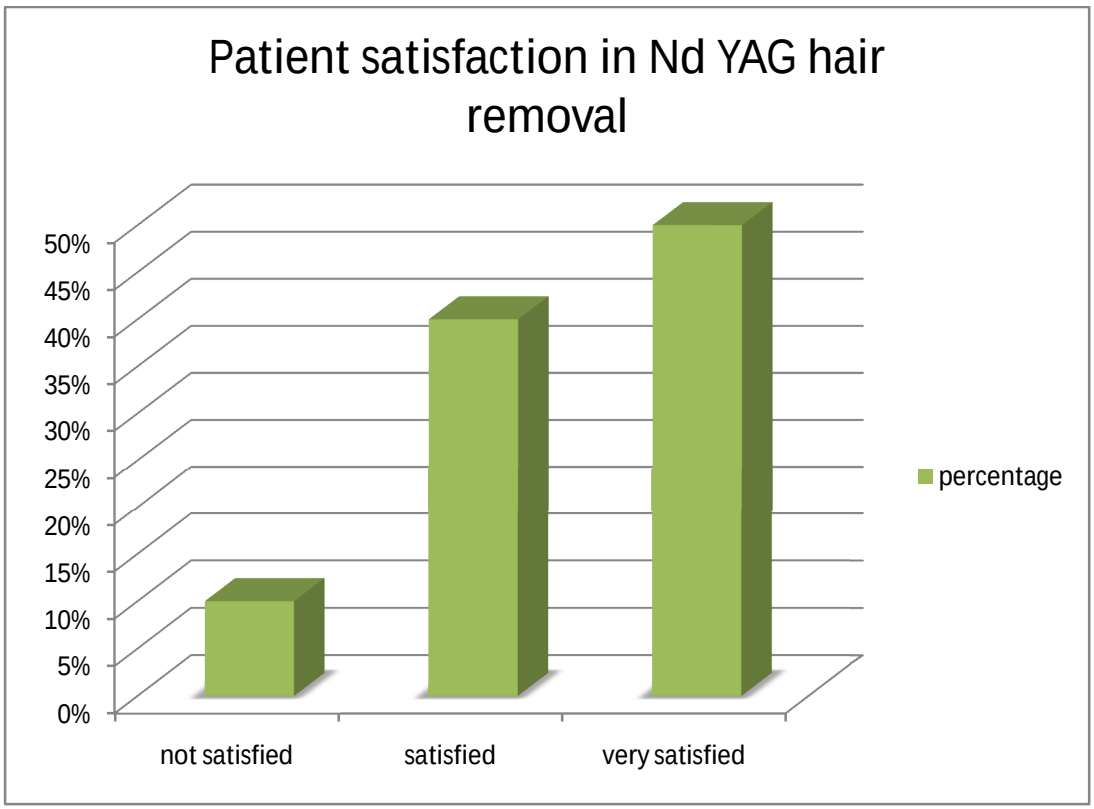

PCOD association in the Nd YAG group
80%
70%
60%
50%
40%
30%
20%
10%
0%
PCOD
nonPCOD
percentage


Skin types of patients in both Nd YAG group & IPL group
10%
40%
50%
type 3
type 4
type 5


Areas of treatment in the Nd YAG group
Percenrage
70
60
50
40
30
20
10
0
40
25
15
20
Upper lip
Upper lip & chin
Upper lip, chin & sides
Chin & sides


Nd YAG laser Treatment response in terms of HRE
80%
70%
60%
50%
40%
30%
20%
10%
0%
mild
moderate
good
excellent
percentage


No. of sessions required for good response
70
60
50
40
30
20
10
0
4th sitting
5th sitting
6th sitting
percentage


Patient satisfaction in Nd YAG hair removal
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
not satisfied
satisfied
very satisfied
percentage

Nd YAG laser treatment:

Before treatment

After 4 sittings of Nd YAG Laser treatment

Before Nd YAG Laser treatment

After 4 sittings of Nd YAG Laser treatment

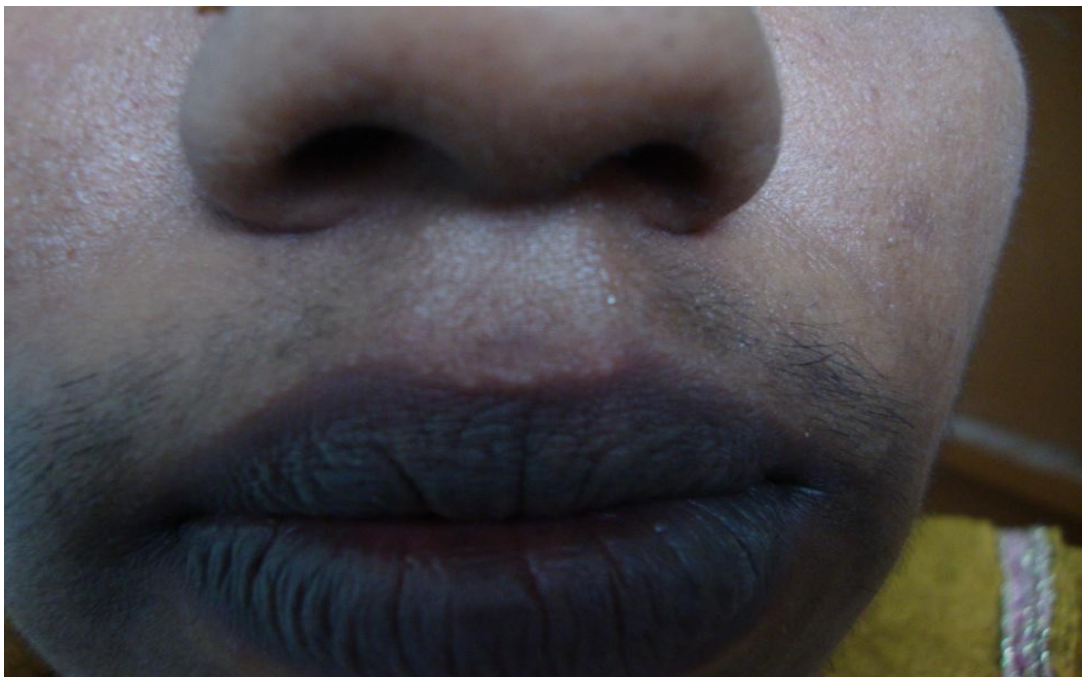
Before Nd YAG Laser

After 5 sittings of treatment

Before Nd YAG laser

After 5 sittings of treatment

Before Nd YAG Laser



After 5 sittings Nd YAG Laser

Before Nd YAG Laser

After 5 sittings of Nd YAG laser

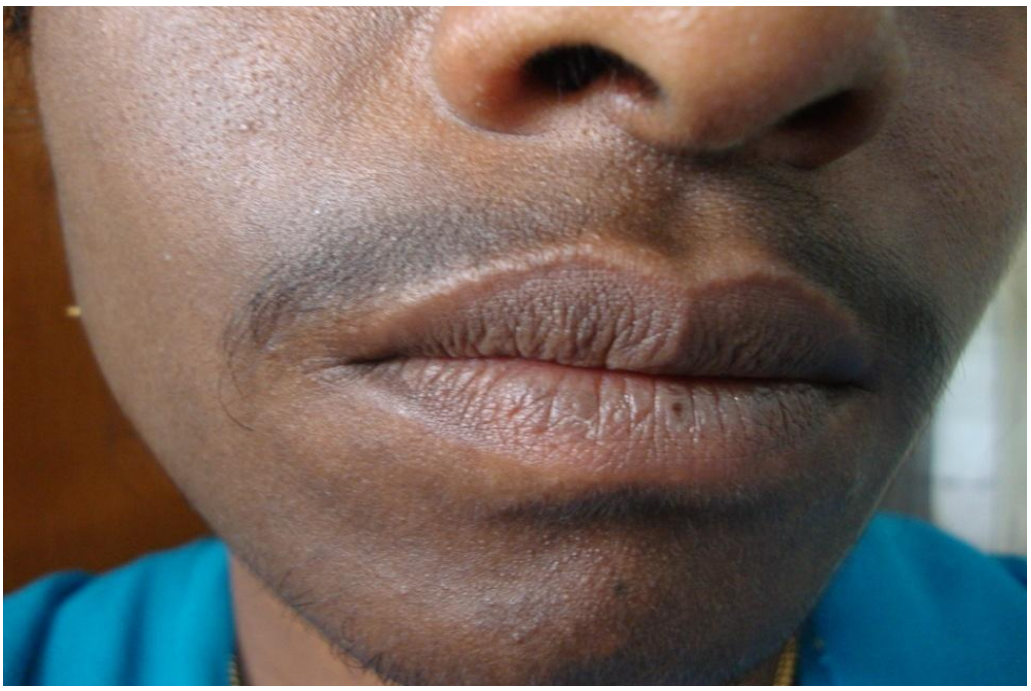
Before Nd YAG Laser

After 5 sittings of Nd YAG Laser

Before Nd YAG Laser

After 6 sittings of Nd YAG Laser

Before Laser treatment



After 5 sittings of Laser treatment

IPL Group:

Table 22 : Age distribution in the IPL study Group

Age group	Frequency	Percentage
18 - 25	9	45.0
26 - 35	6	30.0
36 - 45	5	25.0

Here also, most of the patients were less than thirty five years of age.

Table 23 : Occupations of study patients in the IPL group

Occupation	Frequency	Percentage
Working	8	40.0
House wife	6	30.0
Student	6	30.0

Here all the patients' occupations were almost equally distributed.

Table 24 : Associated factors in the study group

	Frequency	Percentage
PCOS	11	55
Non PCOS	9	45

All the patients were randomly allotted for laser irrespective of the associated factors. So in the IPL group, more number of patients had PCOS. We could not compare the Nd YAG laser & IPL for this reason.

Table 25 : Skin types of study patients in the IPL group

Skin type	Frequency	Percentage
3	2	10.0
4	10	50.0
5	8	40.0

Skin types in both groups were equal in number.

Table 26 : Sites of excessive hair growth in IPL group patients

Site	No. of patients	Percentage
Upper lip	3	15.0
Upper lip & chin	1	5.0
Upper lip, chin & sides	3	15.0
Chin & sides	13	65.0

In this IPL group, more number of patients had hair growth over the chin and sides.

Table 27 : Hair removal efficiency (HRE) in the study population

Degree of improvement	Frequency	Percentage
Mild	0	0
Moderate	3	15
Good	14	70
Excellent	3	15

Among 20 patients in the IPL group, 70% of patients had good response and 15% of patients had an excellent response.

Table 28 : No. of treatment sessions in the study patients who had good response

No. of sessions	No. of patients	Percentage
4	9	47.4
5	6	31.6
6	4	21.0

Table 29: One way ANOVA to find the maximum improvement between sittings in IPL Group.

Sittings	N	Mean HRE	S.D	P value
3	20	43.30	11.904	<0.001
4	20	53.75	8.932	
5	20	50.05	10.154	
6	20	60.85	10.236	

In the IPL group, most of the patients responded after 4 sittings. On further sittings, no improvement in response was seen. This is stastically significant.

Table 30: Mean HRE in different skin types in the IPL group

Skin type	No. of patients	Mean HRE	S.D	P value
3	2	67%	12.727	0.683
4	10	59.8%	10.559	
5	8	60.6%	10.266	

Hair removal efficiency of IPL was almost equal in the different skin types in our study patients.

Table 31: Independent samples t-test to compare the mean improvement between PCOS and non PCOS patients in the IPL Group

	N	Mean HRE	S.D	P value
Without PCOS	9	59.67	9.605	0.653
With PCOS	11	61.82	11.089	

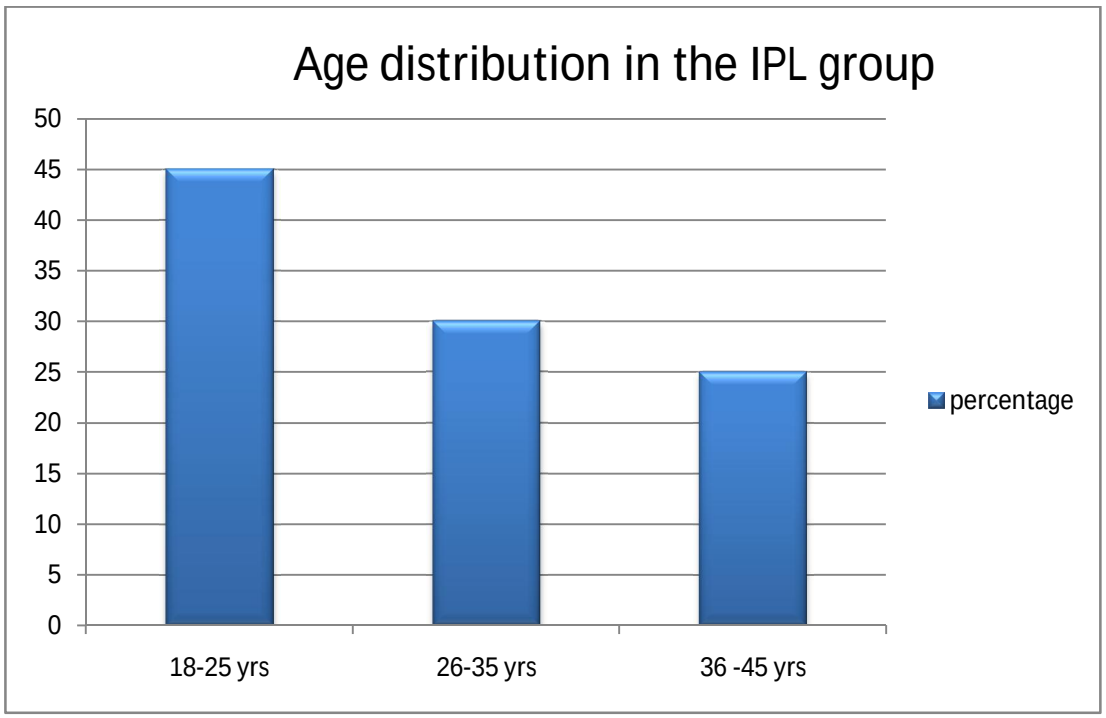
No difference in hair removal efficacy noted in between PCOS and non PCOS patients.

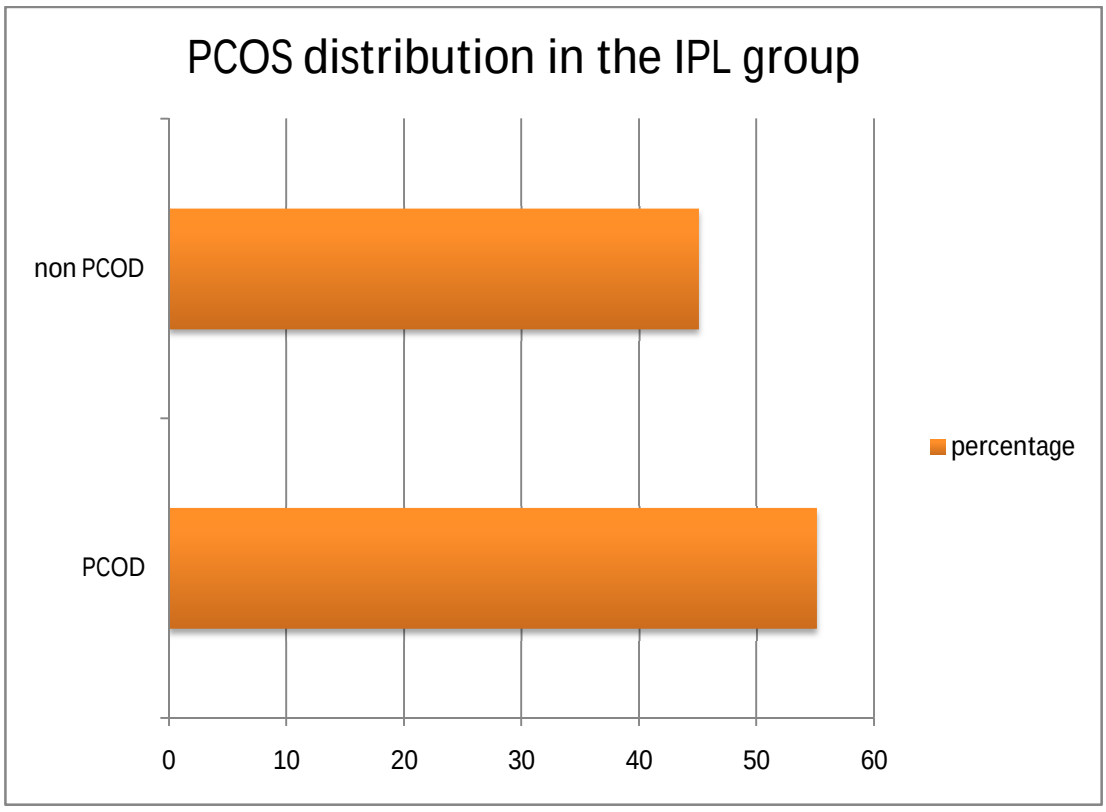
Table 32 : Patient satisfaction for hair removal

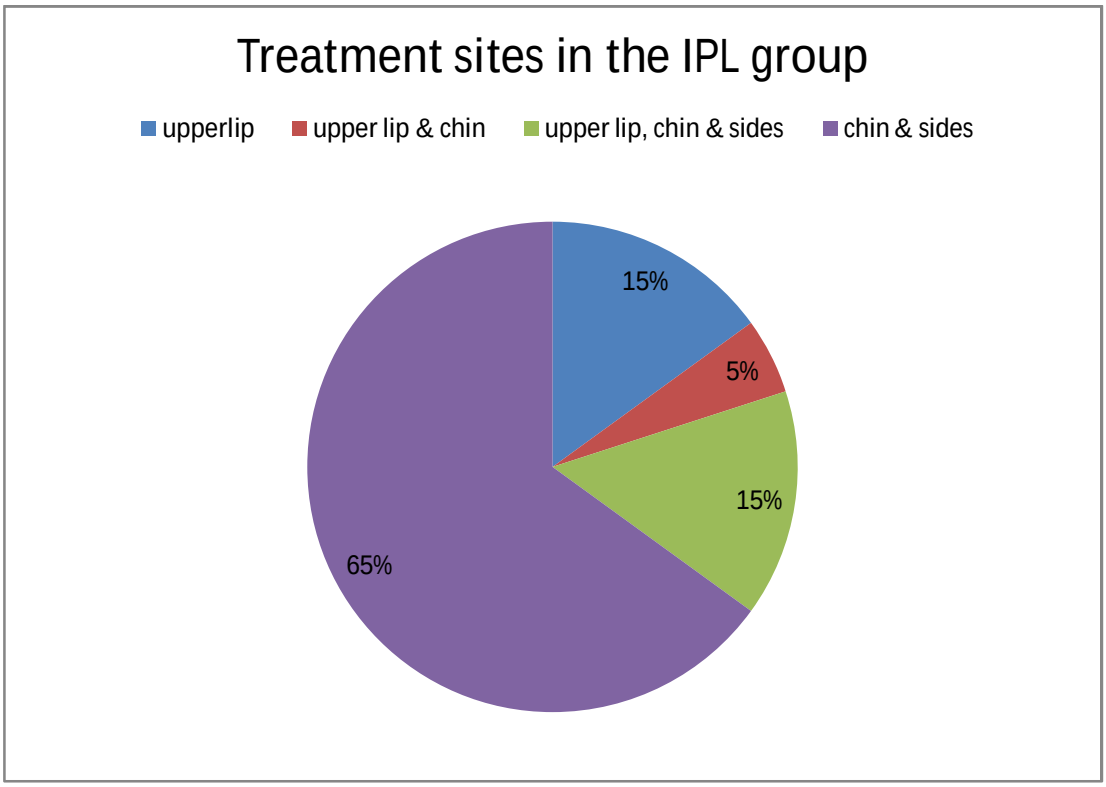
Satisfaction levels	No. of patients	Percentage
Not satisfied	1	5
Satisfied	14	70
Very satisfied	5	25

Among the 20 patients in the IPL group, 70% of the patients were satisfied and 25% of the patients were very satisfied with IPL hair removal. In the IPL group, no side effects were noted.

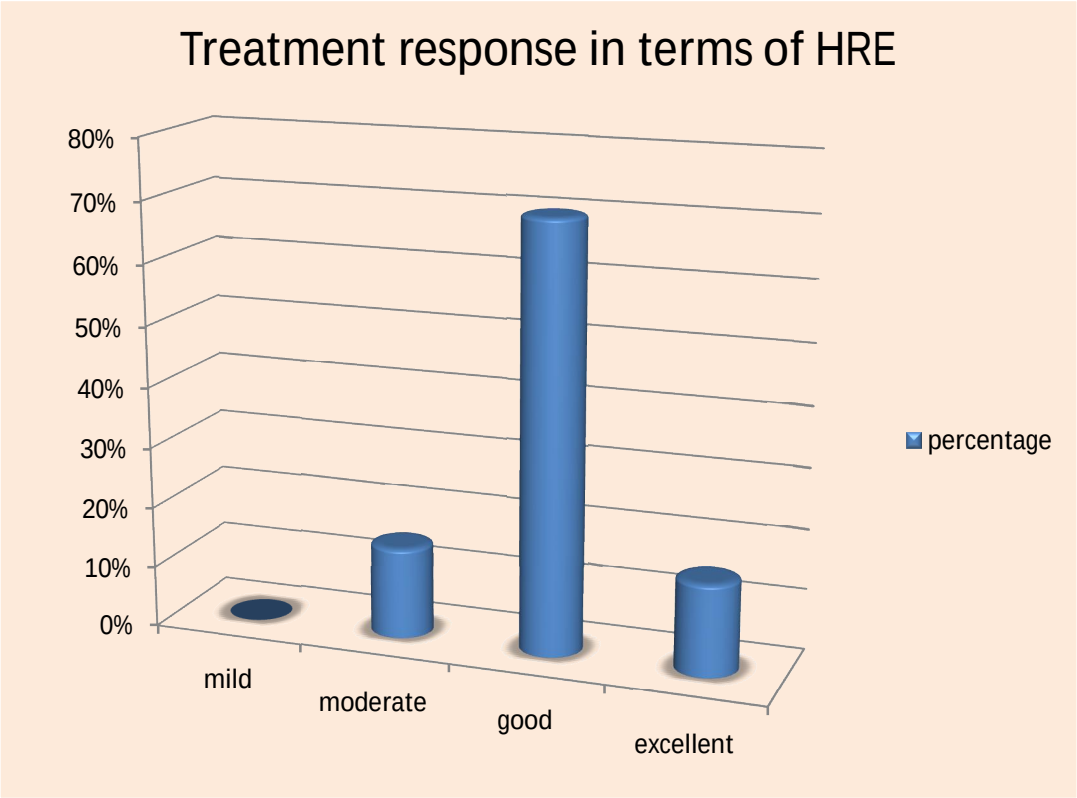
After 6 months, 11 patients came for follow up. Among them, 9 patients maintained their achieved hair removal. But 2 patients developed regrowth of hair.

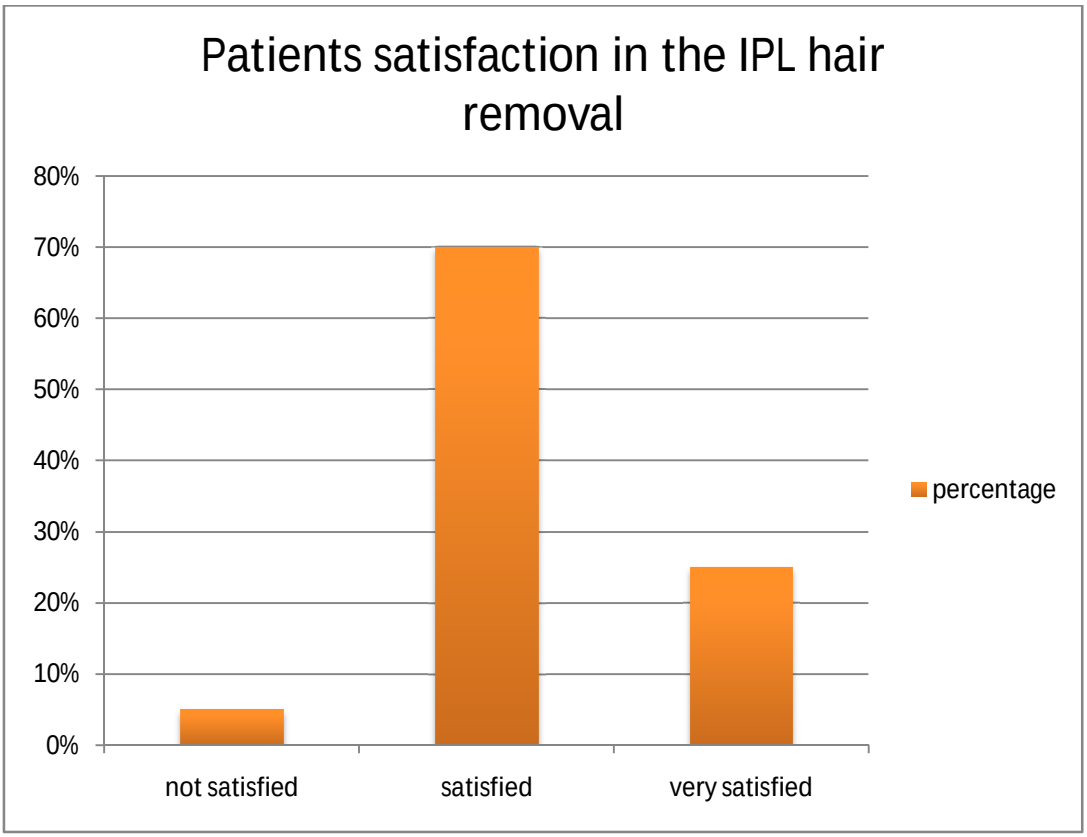

Age distribution in the IPL group
50
45
40
35
30
25
20
15
10
5
0
18-25 yrs
26-35 yrs
36 -45 yrs
percentage


PCOS distribution in the IPL group
non PCOD
PCOD
0
10
20
30
40
50
60
percentage


Treatment sites in the IPL group
upperlip
upper lip & chin
upper lip, chin & sides
chin & sides
15%
5%
15%
65%


No. of sittings required for good response in IPL group
50
45
40
35
30
25
20
15
10
5
0
4th sitting
5th sitting
6th sitting
percentage


Treatment response in terms of HRE
80%
70%
60%
50%
40%
30%
20%
10%
0%
mild
moderate
good
excellent
percentage


Patients satisfaction in the IPL hair removal
80%
70%
60%
50%
40%
30%
20%
10%
0%
not satisfied
satisfied
very satisfied
percentage

IPL HAIR REMOVAL:

Before IPL

After 4 sittings of IPL treatment

Before treatment

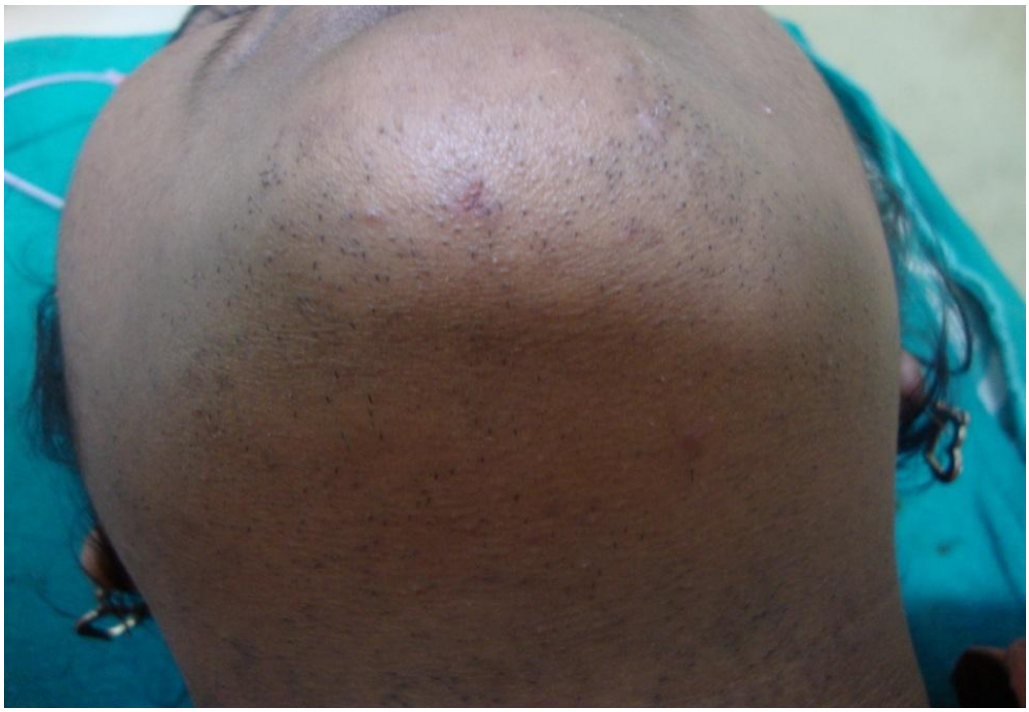
After 4 sittings of IPL

Before IPL

After 4 sittings of IPL

Before IPL

After 5 sittings of IPL



Before treatment

After 4 sittings IPL treatment

Before IPL

After 4 sittings of IPL treatment

Before IPL treatment

After 6 sittings of IPL treatment

Before IPL Procedure

After 5 sittings of IPL

Before IPL procedure

After 5 sittings of IPL

DISCUSSION

Hirsutism is a frequent reason for cosmetic embarrassment, poor self esteem and psychological distress for women world over.

In our study, totally 73 hirsutism patients had been enrolled.

Demographic characters:

Age:

Our patients' age group varied between 14 and 48 years. Mean age was 29.19 years. Most of the patients (72.6%) were in the reproductive age group (15 to 35 years). This feature is supported by Nand Lal Sharma et al.[63] The showed that mean age of hirsutism was 25.84 +/_ 8.30 years, 50% of them were in the age group of 20 - 30 years. Atallah et al[64] and QaziMasood Ahmad et al[65] also supported this feature.

In more than 50% of patients, age of onset of hirsutism was before 20 years.

Duration of disease varied from 1 to 21 years.

Menstrual status:

We noted irregular menstrual cycle in 35 patients (48%) due to PCOS, Cushing's disease and hypothyroidism. This correlated with Tehrani et al who had showed that 54.5% of their patients had menstrual

Androgenetic alopecia:

In this study, 15% of patients had androgenetic alopecia which is supported by Nand Lal Sharma.[63] He described 16% of patients with androgenetic alopecia.

Infertility:

Primary infertility is defined as having the history of trying to conceive for at least one year without success, despite regular sexual intercourse, no use of contraception and no previous pregnancy. Among 41 married patients, 5 patients (12.2%) had no children.

Metabolic syndrome:

We had noted 9.6% hirsutism cases with diabetes mellitus and hypertension. Among 7 patients with diabetes mellitus, 4 patients were above 40 years of age and 3 patients were below 40 years. Magdalena Andries showed 6.6% hirsutism cases with diabetes.[71]

Etiological distribution:

We have noted that among 73 hirsutism patients, 53% had idiopathic hirsutism, which was the most common cause found in our study. Hirsutism with normal ovulatory function and normal circulating androgen concentrations has been defined as idiopathic hirsutism. PCOS was seen in 31.5% of patients. Drugs like cyclosporine and prednisolone were the cause of hirsutism in 4% of cases and 10% had hypothyroidism.

Congenital adrenal hyperplasia and Cushing's disease constitute 1.5% each. Zargar et al found an incidence of 37.3% PCOS cases in 150 Kashmir patients.[72] Qazimasood Ahmad et al showed 80% idiopathic cases in his study on hirsutism in Kashmir which correlates with our study.[65]

Body mass index:

In our study 67% of patients were above normal BMI (>25). This is supported by Atallah et al who showed that 51% of hirsutism patients were obese.[64] As an elevated BMI increases the risk for adverse health sequences, these patients must be followed up regularly.

Ferriman Gallway score:

In this study maximum FG score was 21 which was seen in congenital adrenal hyperplasia. The Average FG score was 15. Most of the patients (73%) had FG score in the range of 9 to 16. All patients were more concerned about facial hair. Qazi Mazood Ahmad et al had seen FG score from 10 to 34 in his study.[65] Face was the most common site. Chest and abdomen were next common sites in their study.

Nd YAG laser hair reduction:

Twenty patients, in the age group of 19 to 44 years, were randomly selected for Nd YAG laser hair reduction. Among them, 6 patients had PCOS.

Skin type:

Most of the patients were skin type IV (50%) and V (40%). Only 10% patients had skin type III. There is no difference in the hair removal efficacy between skin types. This is supported by Ismail.[73]

Sittings:

Most of the patients responded after the 5th sitting. This is statistically significant. Tahir Kamal showed 50% good response in the first sitting.[74] They have used higher energy in the first sitting itself. But in our study energy was started from 25% and gradually increased by 5% for each sitting.

Hair removal efficacy:

In our study 70% patients showed good response (50 to 75% hair reduction) and 20% patients showed excellent response (more than 75% hair reduction) at the end of 6th sitting. Tahir Kamal et al noted good response in 58% cases and excellent response in 20% patients after 6 sittings with fluence. [74] This is comparable with our inference. Goldberg et al showed 59% hair reduction.[75]

Efficacy in PCOS and non PCOS group:

There is no statistically significant difference in hair removal efficacy between PCOS and non PCOS group. Further studies are needed to confirm this finding.

Side effects:

Q switched Nd YAG laser hair reduction resulted in transient erythema and perifollicular edema in 90% patients which resolved spontaneously within 6 hours. Pain was noted in 80% patients. For them local anesthetic was used before procedure in further sittings. Crusting occurred in 10% cases. No pigmentary disturbance or scarring was noted in our study. No patient developed HSV infection after procedure. All these findings are supported by Christopher et al.[76] They had done laser hair removal for 364 patients in various sites and recorded similar side effects.

Follow up:

After 6 months, 10 patients came for follow up. Among them, one patient had hair regrowth of 30 to 40% and 9 patients maintained their hair reduction. Ferraro et al had noted 40 to 65% hair regrowth after hair removal with Nd YAG laser in 480 patients during two years follow up period.[77]

IPL hair removal:

Twenty randomly selected patients, in the age group of 18 to 45 years, were given IPL treatment. Among them, 11 patients had polycystic ovarian syndrome.

Skin type:

Most of our patients had skin type IV and V. There was no change in the efficacy of IPL in the different skin types. This is supported by Ismail. [73] and Khodaeyani et al.[78]

Sittings:

After 4 sittings 45% of patients achieved maximum result. This finding was confirmed by Maya.[79] In her study, IPL was given for type IV and V skin patients and maximum result was achieved after 4 sittings.

Hair removal efficacy:

In this study 70% patients showed good response (>50% hair reduction) and 15% patients showed excellent response (>75% hair reduction) at the end of 6 sittings. This feature was also noted by Tahir kamal et al.[74] He found excellent response in 30% of cases and good response in 62% of cases after the same period.

Efficacy in PCOS and nonPCOS groups:

There was no difference in hair reduction between PCOS patients and non PCOS patients in this study which is also supported by Taylor et al.[80]

Satisfaction level:

In this study, 70% of our patients in the IPL group were satisfied and 25% patients were very satisfied. According to Fodor et al, only 60% patients rated their satisfaction to be good.[81]

Side effects:

None of our patients showed any side effect. Similarly, Harvey jay showed minimal side effects (temporary skin lightening or darkening in 3 patients, acne-like rashes in 6 patients and slight tingling sensations or sensitivity in 2 patients) in 250 hirsutism patients.[82]

Follow up:

After 6 months, 11 cases came for follow-up of which 9 patients maintained their results and 2 patients developed regrowth of hair. Agneta Troilium et al showed long lasting hair reduction in all cases.[83] Small sample size and short follow up may be the reason why we are not able to confirm the finding.

CONCLUSIONS

1. Hirsutism is commonly seen in the age group of 14 to 48 years. Adolescent patients appear to be more concerned about hirsutism as compared to those in the older age group.

2. Acanthosis nigricans (31.5%) was the most common cutaneous finding in our study. Acne was associated in 17.8% patients. Androgenetic alopecia was found in 15% of cases.

3. Infertility was seen in 12.2% case. Diabetes mellitus with hypertension was seen in 9.6% patients and obesity in 28% of patients.

4. Idiopathic hirsutism (53%) was the most common cause in our study. PCOS (31.5%) was the second common cause. Family history was present in 22% cases.

5. Due to PCOS, hypothyroidism and Cushing's syndrome, 48% of patients had menstrual irregularities.

6. Most of the patients (73%) had Ferri Gallwey score in the range of 8 to 16. All patients, however, were more concerned about facial hair than those on other body areas.

7. In Q switched Nd YAG, most of the patients had good response after the 5th sitting. In IPL, majority had good response after the 4th sitting.

8. Good response was seen in 70% of the patients in both NdYAG and IPL groups. Excellent response was seen in 20% of Nd YAG group and 15% of IPL group. There is no difference in hair removal efficacy between PCOS and non PCOS patients.

9. In Q switched Nd YAG and IPL hair removal, there was no difference in efficacy in different skin types.

10. Transient erythema, edema and crusting were the complications observed in Q switched Nd YAG laser hair reduction. No side effects with IPL laser were noted in our study.

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S.N	Age	Age of onset	Mens abn	Marital st	Occupation	Family H/o	Acne	AN	AA	BMI	PCOS	Hypothyroid	Others	HT/DM	Lipid abn	Infertility	FG score	Laser/IPL	Skin type	Initial hair count	Site	HRE in %	Satisfaction	side effect
1	29	12	Y	M	W	Y	Y	Y	Y	32	N	Y	N	Y	Y	N	9							
2	25	23	N	M	W	N	Y	N	N	28	N	N	N	N	N	Y	11							
3	25	20	Y	U	W	Y	N	Y	N	40	Y	N	N	N	N	N	12							
4	40	39	Y	M	HW	N	N	N	Y	30	N	N	D	N	N	N	14							
5	26	25	N	U	W	N	Y	Y	N	24	N	N	N	N	Y	N	12							
6	21	10	N	U	St	N	N	Y	N	26	N	N	N	N	N	N	16							
7	38	15	N	M	HW	N	N	N	N	28	N	N	N	N	N	N	14							
8	27	22	Y	U	W	N	N	N	N	22	N	N	CAH	N	N	N	21							
9	30	28	Y	M	HW	N	N	N	Y	34	N	N	C	N	N	Y	12							
10	29	28	N	M	W	N	N	Y	N	35	N	N	N	N	N	N	18	1	4	28	1	65	3	+
11	15	12	N	U	St	N	N	N	N	24	N	N	N	N	N	N	16							
12	44	25	N	M	W	N	N	N	N	28	N	N	N	N	N	N	16	2	5	18	4	50	2	-
13	29	24	Y	U	W	N	N	Y	N	30	Y	N	N	Y	N	N	19	2	5	26	4	52	2	-
14	15	13	N	U	St	N	N	Y	N	25	N	N	N	N	N	N	9							
15	45	38	N	M	HW	N	N	N	N	27	N	N	N	Y	Y	N	11							
16	27	19	Y	M	W	N	N	N	Y	25	Y	N	N	N	N	N	18							
17	29	23	N	U	W	N	N	Y	N	32	N	N	N	N	Y	N	16							
18	22	19	N	U	St	N	N	N	N	24	N	N	N	N	N	N	14	1	4	12	1	65	2	+
19	23	18	N	U	St	N	N	N	N	22	N	N	N	N	N	N	16	1	4	16	1	60	2	+
20	20	16	Y	U	St	N	N	Y	N	26	Y	N	N	N	N	N	18	1	5	20	3	55	2	+
21	30	25	N	U	W	N	N	Y	N	29	N	N	N	N	N	N	15	1	5	16	2	70	3	+
22	38	15	N	M	HW	N	N	Y	N	26	N	N	N	N	N	N	9							
23	42	14	N	M	HW	N	N	N	N	30	N	N	N	N	N	N	19	2	4	18	4	50	2	-
24	41	20	N	M	W	N	N	N	N	28	N	N	N	N	N	N	16	2	5	21	4	55	2	-
25	22	19	Y	U	St	N	N	N	N	25	Y	N	N	N	N	N	20	2	4	24	3	60	2	-

S.N	Age	Age of onset	Mens abn	Marital st	Occupation	Family H/o	Acne	AN	AA	BMI	PCOS	Hypothyroid	Others	HT/DM	Lipid abn	Infertility	FG score	Laser/IPL	Skin type	Initial hair count	Site	HRE in %	Satisfaction	side effect
26	23	22	N	U	W	N	Y	N	N	25	N	N	N	N	N	N	12	2	4	14	1	55	2	-
27	25	16	N	U	W	N	N	N	N	22	N	N	N	N	N	N	14	2	4	12	4	58	2	-
28	22	18	N	U	St	N	N	N	N	20	N	N	N	N	N	N	11							
29	24	22	Y	M	HW	N	N	N	N	26	Y	N	N	N	N	N	16	1	5	18	1	65	2	+
30	19	10	Y	U	St	N	N	Y	N	29	Y	N	N	N	N	N	16	2	4	25	3	80	3	-
31	30	28	Y	M	HW	N	Y	N	N	28	N	Y	N	N	N	N	18	1	5	24	4	50	1	+
32	22	18	N	U	W	N	Y	N	N	25	N	N	N	N	N	N	10							
33	25	22	Y	M	St	Y	N	N	N	26	Y	N	N	N	N	N	18	2	5	22	4	60	2	-
34	20	16	N	U	St	N	N	Y	N	33	N	N	N	N	N	N	16	2	5	18	4	60	2	-
35	22	21	Y	M	HW	N	Y	Y	N	20	Y	Y	N	N	N	N	11	2	5	20	2	68	3	-
36	19	13	N	U	St	N	N	Y	N	35	N	N	N	N	N	N	12	1	3	16	2	78	3	+
37	16	14	N	U	St	N	Y	N	N	30	N	N	N	N	N	N	10							
38	33	30	Y	M	HW	N	N	Y	Y	32	N	Y	N	N	N	N	18							
39	48	25	Y	M	HW	N	N	N	N	31	Y	N	N	Y	N	N	12							
40	38	33	Y	M	HW	N	N	Y	N	24	N	Y	N	Y	N	N	14							
41	40	30	N	M	HW	N	N	Y	N	32	N	N	N	N	N	N	12							
42	25	18	Y	M	HW	N	N	N	N	28	N	N	D	N	N	N	21							
43	18	12	Y	U	W	Y	N	N	N	27	Y	N	N	N	N	N	16	1	4	22	3	76	3	-
44	45	30	N	M	HW	N	N	N	N	32	N	N	N	N	N	N	14	1	4	18	4	45	1	+
45	42	35	Y	M	HW	Y	N	N	Y	33	N	Y	N	N	N	N	12							
46	18	14	N	U	St	Y	N	N	N	24	N	N	N	N	N	N	11	1	4	18	2	65	3	+
47	19	14	N	U	St	N	N	N	N	23	N	N	N	N	N	N	10	1	4	14	1	68	3	+
48	19	18	Y	M	HW	Y	N	Y	Y	26	Y	N	N	N	N	N	13							
49	33	28	Y	M	W	N	N	N	N	24	Y	N	N	N	N	N	18	2	5	19	4	82	3	-
50	19	16	N	U	St	Y	Y	N	N	19	N	N	N	N	N	N	12	2	3	14	1	76	3	-

S.N	Age	Age of onset	Mens abn	Marital st	Occupation	Family H/o	Acne	AN	AA	BMI	PCOS	Hypothyroid	Others	HT/DM	Lipid abn	Infertility	FG score	Laser/IPL	Skin type	Initial hair count	Site	HRE in %	Satisfaction	side effect
51	36	27	N	M	HW	N	N	Y	N	28	N	N	N	N	N	Y	18	2	5	16	4	58	2	-
52	42	32	N	M	W	N	N	Y	Y	42	N	N	N	Y	Y	Y	19							
53	35	30	N	M	HW	Y	Y	N	N	30	N	N	N	N	Y	N	14							
54	25	17	Y	U	St	N	N	N	N	20	Y	N	N	N	N	N	12	1	3	21	2	66	3	+
55	36	19	N	M	HW	Y	Y	N	N	23	N	N	N	N	N	N	9							
56	20	19	Y	U	St	Y	Y	N	N	24	Y	N	N	N	N	N	11	1	4	18	2	76	3	-
57	18	17	Y	U	St	N	N	N	N	22	Y	N	N	N	N	N	14	1	5	16	4	65	2	+
58	29	19	Y	M	W	Y	N	N	N	24	Y	N	N	N	N	N	18	2	4	14	3	60	2	-
59	32	22	Y	M	HW	N	Y	Y	N	25	Y	N	N	N	N	N	19	2	4	28	4	55	2	-
60	34	27	Y	M	HW	Y	N	N	N	37	Y	Y	N	N	N	N	21	2	4	16	4	45	1	-
61	27	24	N	U	W	N	N	N	N	23	N	N	N	N	N	N	18	1	5	16	3	55	2	+
62	28	25	N	M	HW	Y	N	N	N	31	N	N	N	N	N	N	11	1	4	14	4	65	2	+
63	36	13	N	M	W	N	N	N	N	23	N	N	N	N	N	N	13	1	5	20	1	76	3	+
64	34	21	Y	M	W	N	N	N	N	26	Y	N	N	N	N	N	12	2	4	18	4	60	2	-
65	38	24	N	M	HW	Y	N	N	N	31	N	N	N	N	N	N	11	2	3	21	4	58	2	-
66	22	15	N	U	St	N	N	N	N	21	N	N	N	N	N	N	14	1	5	16	1	64	2	+
67	21	14	Y	U	St	N	N	N	N	24	Y	N	N	N	N	N	10	2	4	22	1	75	3	-
68	18	12	N	U	St	N	N	N	N	26	N	N	N	N	N	N	14	1	4	18	1	72	3	+
69	48	30	Y	M	HW	N	N	N	Y	24	Y	N	N	N	N	Y	12							
70	35	12	Y	M	HW	Y	N	N	N	26	N	Y	N	N	N	N	18							
71	35	14	Y	M	HW	N	N	N	Y	22	N	N	D	N	N	N	15							
72	42	32	Y	M	HW	N	N	Y	N	28	N	Y	N	N	Y	N	17							
73	46	35	Y	M	HW	N	N	N	Y	31	Y	Y	N	Y	Y	N	14							

Key to Master chart

Mens abn	–	Menstrual abnormalities
Marital st	–	Marital status
AN	–	Acanthosis nigricans
AA	–	Androgenetic alopecia
BMI	–	Body mass index
PCOD	–	Polycystic ovarian disease
DM/HT	–	Diabetes mellitus with hypertension
Lipid abn	–	Lipid abnormality
FG score	–	Ferriman Gallway score
Initial hair count	–	No. of hairs per square cm area, done before the first sitting
HRE in %	-	Hair removal efficiency expressed in percentage
Y	-	Yes
N	-	No

Marital status:

M - Married

U - Unmarried

Occupation:

W – Working

HW – House wife

St – Student

Others:

C – Cushings syndrome

D – Drug induced

CAH – Congenital adrenal hyperplasia

Laser / IPL :

1 – Nd YAG Laser

2 – IPL

Skin type:

3 – Fitzpatrick skin type III

4 - Fitzpatrick skin type IV

5 - Fitzpatrick skin type V

Site:

1 – Upper lip

2 - Upper lip & chin

3 - Upper lip, chin & sides

4 – Chin & sides

Satisfaction:

1- Not satisfied

2- Satisfied

3- Very satisfied

ABBREVIATIONS

DHT	-	Dihydrotestosterone
DHEAS	-	Dehydroepiandrosterone sulphate
ACTH	-	Adrenocorticotropic hormone
SHBG	-	Sex hormone binding globulin
CAH	-	Congenital adrenal hyperplasia
BMI	–	Body mass index
LH	-	Luteinizing hormone
FSH	-	Follicle stimulating hormone
TSH	-	Thyroid stimulating hormone
GnRH	-	Gonadotropin releasing hormone
IGF	-	Insulin like growth factor
PCOS	–	Polycystic ovarian syndrome
FG score	–	Ferriman Gallway score
Laser	–	Light Amplification by Stimulated Emission of Radiation
Nd YAG	–	Neodymium Yttrium Aluminium Garnet
IPL	–	Intense Pulsed Light

PROFORMA

Name : Age : Occupation:

Address :

C/o

H/o presenting illness: H/o excessive growth of hair :

Duration :

Site :

Age of onset :

Rate of progression :

H/o voice change -

H/o loss of scalp hair -

H/o seborrhea -

H/o pimples -

H/o milky discharge from breast -

H/o wt gain -

H/o menstrual disturbances -

H/o easy fatiguability -

H/o polyuria/ polydipsia -

H/o abdominal swelling / abd pain -

H/o visual defects -

H/o neck swelling/ tremor -

H/o chest pain -

H/o drug intake -

Past h/o:

Known TB/ bronchial asthma/ hypertension/DM-

Personal h/o:

Menstrual h/o:

Age of menarche : Cycle : flow :

Obstetric h/o:

Family h/o:

G/E Built -

PR- BP- Ht- Wt- BMI

CVS - RS - P/A -

D/E Acne vulgaris -

Acanthosis nigricans -

Alopecia areata -

Moon facies -

Buffalo hump -

Striae -

Hair- distribution -

Ferriman gallway score -

Genital organ & mucosa -

Palms & soles -

Nail -

Investigation:

Blood sugar -

Lipid profile -

Hormone assay- TSH - T3- T4 -

LH - FSH -

Free testosterone -

USG abdomen -

For patients undergoing Nd YAG LASER / IPL :

Sittings	**Energy / Fluence**	**Hair count**	**Side effect**

Specification of Nd YAG laser

Laser type	:	Q switched Nd YAG laser (Akira)
Wavelength	:	1064nm & 532nm
Frequency	:	Upto 5 Hz
Width of pulse	:	20ns
Enegy density	:	<180mj (single pulse) ; <620mj (direct pulse)
Spot diameter	:	1-6mm
Electrical requirement	:	20V / 50 HZ
Cooling System	:	Internal water - to - air cooled
Dimensions (cm)	:	24 x 40 x 18
Net Weight	:	7 kg
Wattage	:	420 w

Specification of IL – 1000

The IL – 1000 marketed by G3 laser Ltd. is the intens pulsed light system used in this study.

Wavelength	:	400 – 1200nm
Fluence	:	Upto 45J
Pulse Duration	:	Upto 15ms
Spot Size	:	8 x 40mm

- In-built water cooling system
- Portable (Less than 20 kgs)
- Single hand Piece with minimum 6 Interchangeable filters
- Filters for HR / SR / VL / PL / AA

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. R. Subha
PG in MDDVL
Madras Medical College, Ch-3

Dear Dr. R. Subha

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Clinical evaluation and therapeutic outcome in Hirsutism" No. 05112010.

The following members of Ethics Committee were present in the meeting held on 24.11.2010 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan, MD	-- Chairperson
2. Prof. J. Mohanasundaram, MD,Ph.D,DNB Dean, Madras Medical College, Chennai -3	-- Deputy Chairman
3. Prof. A. Sundaram, MD Vice Principal , MMC, Chennai -3	-- Member Secretary
4. Prof R. Sathianathan, MD Director, Institute of Psychiatry, MMC, Ch-3	-- Member
5. Prof. R. Nandhini, MD Director, Institute of Pharmacology, Ch-3	-- Member
6. Prof. Pregna B. Dolia , MD Director, Institute of Biochemistry, MMC, Ch-3	-- Member
7. Prof. C. Rajendiran , MD Director, Institute of Internal Medicine, MMC, Ch-3	-- Member
8. Thiru. S. Govindasamy BA.BL	-- Lawyer
9. Tmt. Arnold Soulina	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee